# MEDICAL JOURNAL

## UNIVERSITY OF TORONTO

VOL LIX
NO 1

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# medical journal
## university of toronto

Established December 1923
An Undergraduate Publication

Volume LIX, Number 1
October 1981


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2) Submitted articles should be typed (or legibly printed) double-spaced, single-sided on paper 8 1/2" x 11" (letter size) and they should not be more than 15 to 20 pages (3000 to 4000 words)
3) Illustrations, graphs, diagrams and figures should be put on a separate sheet and identified by Arabic numerals A list of explanatory legends for all the figures and diagrams should be put on another sheet Do not write on the back of photographs
4) References should be checked with the original sources, designated by Arabic numerals in the text, numbered in order of appearance and put on a separate sheet. Each periodical reference should include in order the author's name and initials. Title of the article, Journal name, volume number, page number and year (eg. 1 Rosen HJ Lumbar Invertebral disc surgery, review of 300 cases *Can Med Assoc J. 101 317, 1969)*
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# Mechanism of Electroacupuncture Analgesia (EAA)

**Richard Cheng Ph.D. 8T3**

*Recently, numerous papers have been published on the topic of acupuncture analgesia in the East and the West. More importantly, multidisciplinary research gives a consistent picture showing that acupuncture analgesia is mainly mediated by endorphins (morphine – like peptides produced by our body) and serotonin (a neurotransmitter in the CNS). In the last few years, extensive studies have been carried out in Dr. Bruce Pomeranz's laboratory at the University of Toronto. Many results demonstrate that electroacupuncture (EAA) is one of the best and safest methods for releasing these endogerous chemicals for pain relief. Some of the results are summarized in table A and B.*

**TABLE A**

| Treatment | Action | EAA (4Hz) | EAA (200Hz) |
|---|---|---|---|
| 1 Hypophysectomy * | Deplete pituitary endorphin | ↓ | ND |
| 2 Dexamethasone | Inhibit beta-endorphin release | (partial) ↓ | ND |
| 3 2% saline feeding | Deplete pituitary endorphin | ↓ | ND |
| 4 Type I opiate antagonists<br>(i) Levo-naloxone<br>(ii) Naltrexone<br>(iii) Cyclazocine<br>(iv) Diphrenorphine | Block Type I Opiate Receptors | ↓<br>↓<br>↓<br>↓ | → |
| 5 Dextro-naloxone | inactive isomer | → | ND |
| 6 D-Leucine and D-Phenylalanine | may enhance endorphins | ↑ | ND |
| 7 Two EA treatments at 3 hr apart | Accumulative effect | ↑ | ND |
| 8 Morphine-addicted mice during withdrawl | addiction | ↑ | ND |
| 9 PCPA | deplete 5-HT | → | ↓ |
| 10 Cinanserin | 5--HT receptor blocker | ND | ↓ |
| 11 5-HTP | 5-HT precursor | → | ↑ |

key: ↓ reduce EAA; ↑ increase EAA; → no effect on EAA.
* from my M.Sc. thesis ND = not done

**TABLE B**

Summary of monoaminergic drugs on EAA at 200 Hz.

| Drug | Net Functional Change | | | EAA |
|---|---|---|---|---|
| 1. TBZ | S↓ | D↓ | N↓ | ↓* |
| 2. PCPA | S↓ | | | ↓* |
| 3. AMPT | | D↓ | N↓ | ↓* |
| 4. Disulfiram | S↑ | | N↓ | ↑* |
| 5. TBZ + 5HTP | S↑ | D↓ | N↓ | ↑* |
| 6. TBZ + L-DOPA | S↓ | D↑ | N↓ | R (Partial) |
| 7. PCPA + 5HTP | NORMAL | | | R* |
| 8. AMPT + L-DOPA (1 hr) | | D↑ | N↓ | R |
| 9. AMPT + L-DOPA (2 hrs.) | | D↓ | N↑ | R (Partial) |
| 10. 5HTP | S↑ | | | ↑* |
| 11. L-DOPA | | D↑ | | ↑ |
| 12. Probenecid | S↑ | D↑ | | ↑* |
| 13. Apomorphine | | D↑ | | ↓ |
| 14. Cinanserin | S↓ | | | ↓* |
| 15. Haloperidol | | D↓ | | ↓ |
| 16. Pimozide | | D↓ | | → |
| 17. Yohimbine | | | N↓ | ↓ |

S - Serotonin, D - Dopamine, N - Norepinephrine
R - Recovery of EAA after replacement drugs.
* only the data related to serotonin gave consistent EAA results.

By combining the summarized results in table A and B with that of the literature, the author proposes an intricate system for the mechanisms of EAA (see Figure 1). Namely, electroacupuncture (EA) at 4 Hz may stimulate the sensory receptor in the deep muscle causing the midbrain PAG to release enkephalins which activates the dorsal raphe nucleus or nucleus raphe magnus and nucleus reticularis magnocellularis (Rmc) to send down descending inhibitions along the DLF to the spinal cord. The raphe-DLF system may be mediated by the neurotransmitter, serotonin (while the Rmc-DLF systems may be mediated by noradrenaline). In parallel, EA (4 Hz) may stimulate the beta-endorphin neurons in the hypothalamus. These hypothalamic neurons project to different areas of the brain (e.g. PAG, periventricular nucleus of thalamus, accumbens, amygdala) and may release beta-endorphin for pain-relief. Additionally, the hypothalamus may also produce a releasing factor to stimulate the release of pituitary endorphins and ACTH. The pituitary endorphins may be released into the blood circulation, or may backflow into the CSF (the endorphins in the circulation have to pass through the blood-brain barrier to bind to the opiate receptors for analgesia). If the acupuncture points and the painful areas are in the same segmental levels, EA (4 Hz) may also directly stimulate the endorphins in the spinal cord. The spinal cord endorphins may presynaptically inhibit the release of the neurotransmitters from small primary afferent fibres and thus block the pain message (nociception). Furthermore, high frequency (200 Hz) EAA may activate sensory nerves and directly stimulate the DLF-serotonin descending inhibitory systems, by-passing the PAG-endorphin systems.

In summary, low-frequency (4 Hz) EA may stimulate the release of endorphins in the spinal cord, midbrain, hypothalamus and pituitary for pain-relief, while high frequency (200 Hz) may directly stimulate the DLF-inhibitory systems, thus avoiding the endorphin links. The details and evidences for this intricate system of EAA are discussed according to the sites of EA actions as follows:

## Possible anatomical sites of EAA mechanisms:

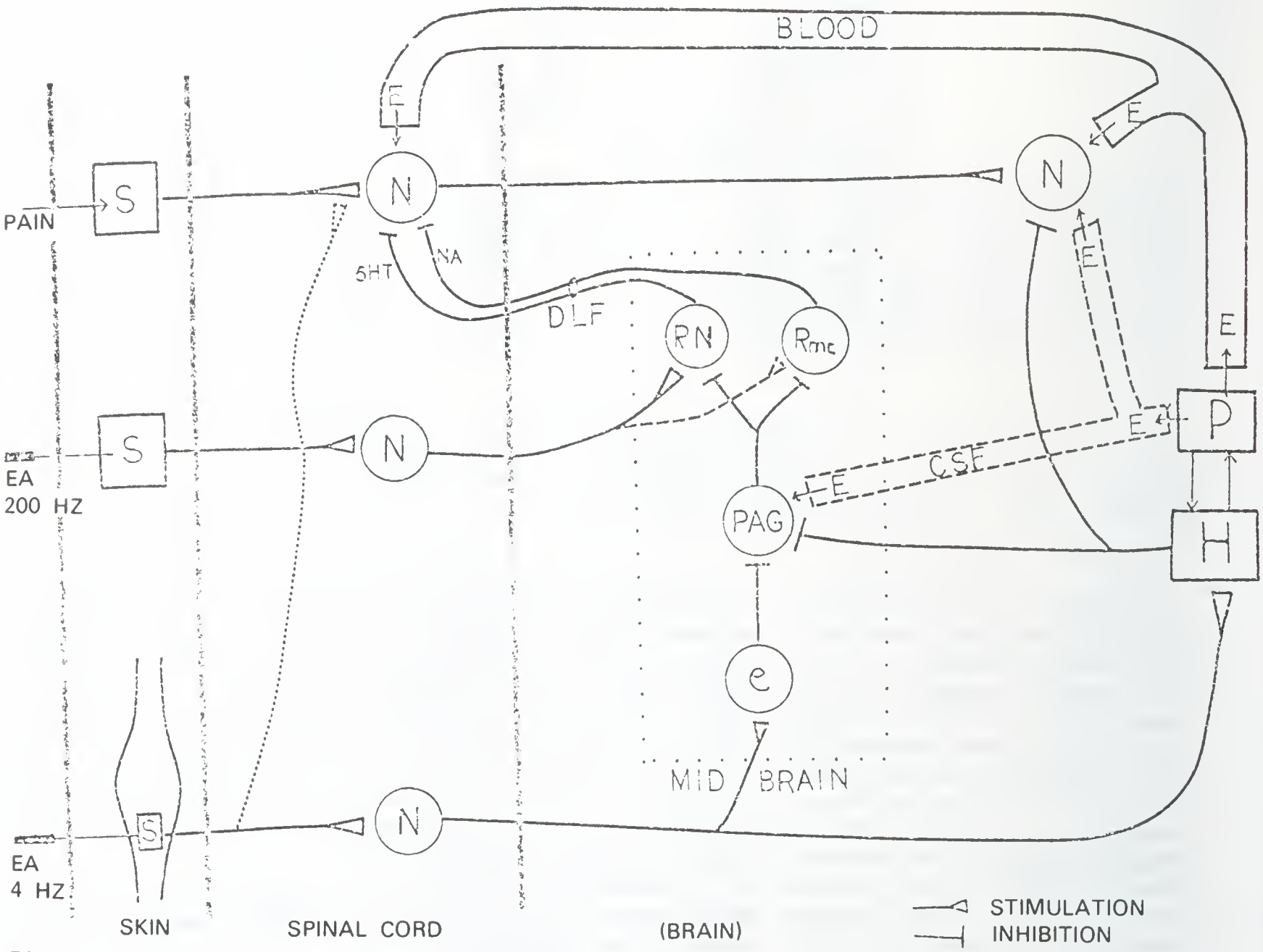
Collectively, the acupuncture-endorphin hypothesis may be divided into three levels of analgesia:

(A) **Local:** Such analgesia is probably localized over a small area due to a segmental release of endorphins in the spinal cord according to the area of stimulation. Several lines of evidence have suggested that substance P is the neurotransmitter at the terminals of small primary afferent (Ad and C) fibres responsible for pain transmission. Immunohistochemical

analysis has revealed that enkephalin and substance P neurons have an intimate spatial relationship in the spinal cord and brain stem. Depleting substance P by capsaicin produces analgesia and enhances morphine analgesia. Moreover, it has been demonstrated that endorphins are able to suppress the release of substance P. Consequently, the release of endorphins may presynaptically inhibit noxious input. If substance P is the neurotransmitter that plays a role in the perception of pain, the closely associated enkephalinergic neurons will inhibit noxious input rapidly, since it takes about 1 msec. for the released endorphin to cross the synapse and produce an effect.

Clinically, it is often observed that EA produces greater analgesia if the painful area and the acupuncture points are at the same specific segment. Sjolund and colleagues (1977) demonstrated that tran-

FIG. 1




EA: ELECTROACUPUNCTURE
S: SENSORY RECEPTOR
N: INTERNEURON
C: ENKEPHALINERGIC NEURON
E: ENDORPHIN (B-ENDORPHIN or DYNORPHIN)
NA: NORADRENALINE

H: HYPOTHALAMUS
P: PITUITARY
PAG: PERIAQUEDUCTAL GRAY
RN: RAPHE NUCLEUS
RMC: RETICULAR MAGNOCELLAR NUCLEUS
DLF: DORSOLATERAL FUNICULUS

Figure 1: At low frequency (4Hz), EA may stimulate the midbrain (PAG) to release enkephalins which will indirectly stimulate the raphe nucleus (RN) and/or reticular magnocellular nucleus (Rmc) to send a descending inhibition on the spinal cord pain cells. Serotonin and noradrenaline are probably the neurotransmitters involved in the RN and Rmc systems respectively. In parallel, EA may also stimulate the hypothalamus and pituitary to release beta-endorphin or dynorphin. The pituitary endorphins may either go through the blood-brain barrier or backflow to the hypothalamus or CSF and bind to the opiate receptors in the spinal cord and the brain. In addition, low frequency (4Hz) EA may cause the segmental release of endorphins from the spinal cord interneurons and bind to the opiate receitors in the pain transmission cells.
High frequency (200Hz) EA appears to stimulate directly the RN and descending inhibitory systems, bypassing the endorphin system.

scutaneous electroacupuncture (TEA) treatment upon the lumbar area elevates endorphin levels in human lumbar CSF but TEA at a distal site (Hoku - First Interosseus muscle on the hand) does not change the endorphin level in the human lumbar CSF. Terenius and his colleagues (personal communication) have found that low-frequency transcutaneous electroacupuncture increases endorphin levels in human lumbar CSF but that high frequency EA has no effect on CSF endorphins, suggesting that low-frequency EA is a better technique for releasing spinal endorphins for segmental pain relief. Application of opiates or endorphin on the spinal cord reduces the noxious output of lamina V neurons or produces a behaviorally-defined analgesia in rabbits, rats, cats and primates. Systemic injection of naloxone also antagonized the EA suppression of the noxious output of lamina V in cats. Recently, Peets and Pomeranz demonstrated that microinjection of naloxone in the spinal cord abolished transcutaneous nerve stimulation analgesia in rats (personal communication).

(B) **Regional:** Regional analgesia may be due to the release of enkephalins in the midbrain. These enkephalins stimulate the raphe nucleus, promoting descending inhibition to noxious input via the dorsolateral fasciculus to spinal regions. It has been advocated that the existence of regional mapping in the midbrain and that stimulation of certain midbrain areas will cause analgesia in only part of the body. Acupuncture may exert a similar regional analgesic effect. For example, low-frequency (4 Hz) EA may activate the periaqueductal gray (PAG) to release enkephalins which can act on the brainstem area. It has been demonstrated that the nucleus raphe magnus releases serotonin while the nucleus reticularis magnocellulis releases noradrenaline through the DLF. It should be noted in relation to EAA that the micro-injection of naloxone into PAG partly reversed EAA, while lesioning of the DLF and raphe nucleus abolished EAA. Recent evidence suggests that microinjection of morphine into the PAG evokes the release of serotonin from the spinal cord. Consequently, the inhibition of the spinal sensory system by the PAG opiate complex associated with antinociception appears to be mediated by the joint excitation of serotonin and noradrenaline terminals in the spinal cord via the DLF. In the PAG, the release of enkephalin-opiate receptor binding receptors on the neurons that inhibit the nucleus reticularis magnocellularis (NRM) or nucleus raphe magnus (NRM). This enkephalinopiate receptor binding inhibits the neuronal firing, promoting descending inhibitory systems.

At high-frequency stimulation, EA probably stimulates the raphe neurons, also producing a descending inhibition to the spinal cord through the dorsolateral fasciculus and this seems to be mediated by serotonin. Since high-frequency EAA is not blocked by naloxone and is only partially reversed by depleting serotonin, other descending systems such as the nucleus reticularis magnocellularis may also be stimulated. From other studies on analgesia, it is most probable that noradrenaline is involved in this system. Thus high-frequency EA may directly stimulate descending inhibitory neurons by-passing the endorphin-link mapping in the PAG.

(C) **General:** EA was shown to exert a segmental and non-segmental effect. Chapman *et al.* (1975) observed that non-segmental EA (Hoku on the hand) produced a 40% analgesia on tooth-pain, while segmental EA (cheek area) produced 185% analgesia. However, it was demonstrated by Chiang *et al* (1973) that the non-segmental EA stimulation on Hoku caused an increase in pain threshold over the entire body without significant differences among different body areas. This non-segmental EAA is quite different from the local segmental pain relief and may be mainly due to brain and/or pituitary endorphin release. Therefore, in a parallel system, especially at low-frequency electrical current, EA may stimulate the hypothalamus and pituitary to release beta-endorphin and ACTH, which may serve both pain-relieving and therapeutic functions. Pituitary endorphins can either be released into the blood stream or directly into the CSF. Evidence for pituitary-brain transport has been demonstrated by Mezey and colleagues (1978). The backflow of pituitary hormones tc the hypothalamus may be partly vascular via the hypothalamic-hypophyseal portal system and to other brain areas via the CSF. Hypothalamic beta-endorphin neurons in the arcuate nucleus are also shown to project to the PAG, locus coeruleus, reticular formation, medial amygdala, accumbens and the periventricular nucleus of thalamus. It is still not clear, however, whether EA has a direct effect on stimulation of these long projecting beta-endorphin neurons of the hypothalamus. If indeed, EA has a strong effect in releasing hypothalamic and pituitary endorphins, the analgesic effect will probably cover the whole body and be of long duration, since these high-molecular weight endorphins can reach a wide variety of brain regions by passing through the blood-brain barrier or via the CSF in eliciting general analgesia. Recently, a novel endorphin, dynorphin, was found in the posterior pituitary gland (Goldstein *et al*, 1979) while beta-endorphin was found in the anterior pituitary. Further research is required to find out whether dynorphin is involved in EA or stress-induced analgesia. □

## References:

Extensive references available upon request.

# Hypertrophic Cardiomyopathy:

## Current Concepts and Areas of Controversy

**Anne Curtis 8T2**

*Hypertrophic cardiomyopathy is an idiopathic disease of heart muscle, comprising asymmetric septal hypertrophy without dilation and myocardial disarray. Researchers believe either systolic obstruction to left ventricular outflow or decreased diastolic compliance (with impaired left ventricular filling) is the main pathophysiologic mechanism. Despite an HLA-linked, autosomal dominant form, most cases are sporadic, developing after prolonged hypertension. Catecholamines may play a role in utero in causing this disease. Hyperthyroidism is more prevalent than expected in these patients. Both myocardial dysplasia and coexisting structural lesions may be caused by an in utero cardiotoxic agent.*

*Annual mortality is 3 to 4%, usually from sudden death. Patients may require vigorous treatment with anti-arrhythymics. Ventriculomyotomy will correct the Venturi effect produced by the asymmetric septal hypertrophy. Other researchers believe in high-dose beta-blockage. Verapamil, a slow-channel calcium antagonist, provides more symptomatic improvement than propranolol, although both drugs have similar effects on exercise tolerance.*

*Hypertrophic cardiomyopathy* is an idiopathic primary heart muscle disease characterized by marked left ventricular hypertrophy — particularly of the ventricular septum — without dilatation. Myocardial disarray — that is, hypertrophied, disorganized, bizarrely-shaped muscle cells — is also typical. In an analysis of over 1600 patients, Maron and Epstein[1] found that the specificities of asymmetric septal hypertrophy, marked septal disorganization, and systolic anterior motion of the anterior mitral leaflet, were 90, 93 and 97%, respectively, in making a diagnosis of hypertrophic cardiomyopathy. Extensive septal disorganization was 90% sensitive for this disease.

### Pathophysiology

Wigle *et al*[2] consider systolic obstruction to left ventricular (L.V.) outflow to be the most important pathophysiologic mechanism producing symptoms in hypertrophic cardiomyopathy. Goodwin[3], however, believes that decreased L.V. compliance in diastole, with resultant impaired filling, is the fundamental problem. This is one of the major unresolved conflicts in this field today. As will be shown later in the review, whether one considers the basic dysfunction to be systolic or diastolic has many ramifications — it determines the rationale for treatment and the prognostic indicators used in different centres.

Goodwin supports his view with that of Swan *et al*[4] that disappearance of systolic obstruction is usually a sign of increasing severity of disease. He also points to observations by Maron *et al*[5] that when maloriented myocardial fibres are widely distributed in the L.V. apex or posterior wall, obstruction is absent, and that myofibrillar disarray is confined to the septum in patients with obstruction, which implies that disease may be very widespread, yet non-obstructive. Conversely, Rakowski *et al*[6] showed that localized subaortic hypertrophy can be strategically located to provoke L.V. outflow obstruction, and yet not extensive enough to alter ventricular filling characteristics.

Wigle cites Adelman *et al*[7] and their experience that patients with latent muscular subaortic stenosis have normal L.V. end-diastolic pressure and compliance, so their symptoms are presumably related to provokable L.V. outflow obstruction. They theorized that this obstruction and the associated mitral regurgitation are the causes of symptoms early in hypertrophic cardiomyopathy, but that in the end stage, the underlying myocardial disorder primarily determines symptoms. This is compatible with Goodwin's observation that progression of disease may be associated with spontaneous loss of L.V. outflow obstruction. In the end stage, the outflow gradient may be lost because the failing heart dilates and contracts less forcefully.

Outflow obstruction, when present, is dynamic, and depends on L.V. end-diastolic volume, myocardial contractility, and the systolic pressure distending the L.V. outflow tract. Isoproterenol, digitalis, nitroglycerine, amyl nitrate, the Valsalva manoeuvre, exercise and tachycardia all *worsen* obstruction. Phenylephrine, squatting, isometric handgrip exercise, leg elevation to increase venous return, and an increase in blood volume, all *lessen* obstruction.

## Clinical Features

Hypertrophic cardiomyopathy presents with dyspnea, angina, syncope, dizziness and L.V. failure. The JVP shows a prominent "a" wave, the peripheral pulse has a rapid upstroke, and the apical pulse is doubled or tripled. There is an S4, and perhaps an S3 and paradoxically-split S2. The systolic ejection murmur is best heard along the left sternal border and at the apex; mitral regurgitation may also be present.

The diagnosis is supported by an EKG showing L.V. hypertrophy and deep, broad Q waves due to septal hypertrophy. Echocardiography usually shows a ventricular septum 1.3 times as thick as the posterior free wall. There is also systolic anterior motion of the anterior mitral leaflet.

Clark *et al*[8] used echocardiography to study families with hypertrophic cardiomyopathy, and showed that 50% of first-degree relatives have septal involvement, though this is often asymptomatic. The disease is transmitted as an autosomal dominant with a high degree of penetrance. Darsee *et al*[9] studied HLA linkage and divided patients into two subgroups: those with an inherited, non-hypertensive form linked to the HLA loci on chromosome 6, and those with a sporadic non-HLA linked form associated with severe systemic hypertension. Petrin *et al*[10], in a 6-year retrospective study in a large community hospital, found that 39 of 46 patients were over 50, 32 were women, and 25 of these had significant hypertension. They concluded that, in a community — as opposed to a large referral centre — the disease is usually non-familial, predominates in elderly females, and tends to be acquired after a lengthy period of hypertension.

## Pathogenesis

Perloff[11] discusses the *catecholamine hypothesis* of the pathogenesis of hypertrophic cardiomyopathy, in which norepinephrine from the neural crest acts on supersensitive fetal myocardial adrenergic receptors to initiate myocardial cellular disarray. This disarray leads to isometric contraction, so that fetal disproportionate spetal thickness does not regress, and indeed, may progress to clinically overt disease. This theory is supported by reports that infants of diabetic mothers and syndromes such as lentiginosis, von Recklinghausen's disease and Friedreich's ataxia — which all have disorders of catecholamines and the sympathetic nervous system — have all been associated with cases of hypertrophic cardiomyopathy.

Bell *et al*[12] noted a 13% incidence of *hyperthyroidism* in patients with hypertrophic cardiomyopathy, and felt that the hyperthyroidism precipitated or exacerbated the disease where there was an incipient myocardial abnormality. Olsen *et al*[13] had previously shown that thyroxine given to pregnant rats could produce changes in the offspring's myocardial ultrastructure similar to the disease. The significance of this data is not clear, since in humans, the patients themselves had hyperthyroidism, where as thyroxine given to adult rats produced no myocardial changes. Bell believes that the thyroxine acts on the heart directly, rather than by inducing catecholamine hypersensitivity, as Perloff suggested.

Somerville and Becu[14] point out that simple *structural lesions* — such as secundum ASD, small VSD, persistent ductus — may be associated with hypertrophic cardiomyopathy. The incidence of this association was underestimated in the past because it was not specifically looked for. The cardiomyopathy must be obvious for its manifestations to overshadow those of the structural lesion. Clues are an atypical clinical course, cardiomegaly, L.V. hypertrophy (where the lesion affects the heart's right side), QS patterns, left anterior hemiblock, and deep septal T-wave inversion. Myocardial dysplasia is also associated with these lesions, and may be a non-specific cardiac response to damage, whether from rubella, vitaminosis D or E, anticonvulsant drugs, or the other previously — mentioned syndromes. Thus the structural lesion and the dysplasia may be due to the same underlying cardiotoxic agent, rather than one causing the other.

## Natural History and Prognosis

Maron and Epstein[15] point out that our understanding of the natural history of the disease is flawed because only one comprehensive study, that of Frank and Braunwald[16] was done before the widespread use of propranolol and myotomy, designed to modify the course. At the time of that study, echocardiography — which can discover patients at the benign end of the disease spectrum (few or no symptoms) — was not yet in use. Frank and Braunwald and Parker[17]alike felt that the disease was only slowly progressive usually, with 25% of patients deteriorating or dying. Adelman *et al*[1] reported that 75% of untreated patients deteriorated or died, and 66% ended up in functional class III or IV. The course is generally accepted to be quite variable.

Each year, 3 to 4% of the patients die prematurely from the disease. Shah *et al*[18] found that 87% of these deaths were sudden, presumably from arrhythmia, and the rest died from progressive congestive heart failure. There has been considerable interest in finding *predictors of sudden death*.

McKenna *et al*[19] found that sudden death could best be predicted by a combination of age under 14, syncope at diagnosis, severe dyspnea at the last follow-up, and a family history of hypertrophic cardiomyopathy and sudden death; false negatives were 30%, false positives, 27%. This can be stratified according to age. Patients diagnosed in childhood tended to be asymptomatic, but with an unfavourable family history and a 5.9% annual mortality. In those 15 to 45 at diagnosis, syncope was the only prognostic feature; annual mortality rate was 2.5%. Dyspnea and exertional chest pain were danger signs in patients aged 45 to 60, who had a 2.6% annual mortality. The history at the time of diagnosis, and changes in symptoms during follow-up, were better prognostic indicators than EKG or hemodynamic measurements.

Orinius[20], in contrast, did not find initial symptoms significant, and recommended these criteria for long-term prophylaxis against ventricular fibrillation (and hence, sudden death): an absent Q in lead III, and cardiac enlargement on chest X-ray at the initial examination. In his series, this combination was present in 75% of the deaths and 13% of survivors. It is crucial to remember that sudden death may occur at any age or in either sex, whether or not symptoms have been present. Thus, many authors now recommend Holter monitoring, even of asymptomatic patients, and aggressive treatment of any arrhythmias discovered.

Studies have been done to ascertain *pathologic abnormalities* underlying sudden death. James and Marshall[21] found abnormal fibrosis of the sinus node, associated with or independent of a narrowed sinus nodal artery, in 12 of 22 patients. 13 of the 22 hearts had obvious fragmentation of the AV node or bundle of His. Other important findings included narrowing of many small coronary arteries in the septum, and deep clefts from the endocardium into the septum. Rowland *et all*[22] have demonstrated accessory pathways of hypertrophic cardiomyopathy tissue. Atrial fibrillation is relatively uncommon, but poorly tolerated and indicates advanced disease. Progressive L.V. dysfunction and increasing left atrial enlargement may predispose to atrial fibrillation; alternatively, the atrial "kick" may be required in patients with decreased L.V. compliance — without it, L.V. filling may be inadequate and decompensation may occur.

Maron *et al*[23] found postmortem evidence of past transmural myocardial infarction in 7 patients with normal extramural coronary arteries, but significant narrowing of intramural arteries by intimal and medial thickening. However, angina, syncope, deep septal Q waves and sudden death may be present in hypertrophic cardiomyopathy alone, without myocardial infarction.

## Treatment

The three major forms of treatment are beta-adrenergic blockers, calcium-channel blockers, and ventriculomyotomy-myectomy.

*Surgery* is best for those with outflow obstruction at rest, since it widens the L.V. outflow tract so that the anterior mitral leaflet is not pulled forward against the hypertrophied septum (the Venturi effect). Once systolic anterior motion of the mitral leaflet is abolished, L.V. outflow obstruction and mitral regurgitation are decreased or eradicated (Wigle *et al*[24]).

Opinions vary about the adequacy of *beta-adrenergic blockage* in patients with obstruction at rest. Wigle believes that it neither controls symptoms nor prevents sudden death, and may, in fact, increase both. Frank *et al*[25] argue that none of *their* 22 patients died over a mean 5-year period, on an average propranolol dose of 462 mg per day. Moreover, they had significant symptomatic improvement, and while 50% had potentially life-threatening arrhythmias, none died. Some patients had a decreased frequency and severity of syncope, presyncope and palpitations, and an end to ventricular arrhythmias, with an increase in the propranolol dose. Compare this to the 3-4% annual mortality from the disease, and an up to 14% mortality rate from surgery; without aggressive medical therapy, further late deaths occur.

Rosing *et al*[26] note that the left ventricle in hypertrophic cardiomyopathy is hypercontractile, with end-systolic apical cavity obliteration and increased ejection fraction. Increased myocardial cell *calcium* could increase contractility; they cite studies on hereditary cardiomyopathy in Syrian hamsters which had an increased uptake and content of calcium in the myocardium. *Verapamil*, by impairing calcium inward flux across membranes, prevented metabolic and anatomic abnormalities in the hamsters. Verapamil IV had a dose-related effect in decreasing basal and provoked L.V. outflow tract gradients. This must have been due to an increase in outflow tract size, since cardiac output remained the same, or rose. Verapamil orally increased exercise tolerance by 15% or more, and/or improved symptoms (beyond the placebo effect) in 14 of 19 patients. 9 patients felt best on verapamil, 7 on placebo, and 3 on propranolol. Exercise capacity was, on average, equal with verapamil or propranolol — however, 3 patients got worse on propranolol, compared to none on verapamil.

It is important to note that patients on propranolol may have more improvement in exercise tolerance than in symptoms. Propranolol may delay the onset of chest pain, dyspnea and lightheadedness during exercise, but it also causes general fatique, so the patients do not subjectively feel better. A point to consider with slow-channel calcium antagonists in that a significant decrease in systemic resistance may increase the outflow gradient. Hence, potent vasodilators such as nifedipine may be contraindicated.

Non-obstructive hypertphic cardiomyopathy is due to impaired diastolic ventricular filling. Agents such as verapamil, capable of decreasing elevated pulmonary wedge and L.V. end-diastolic pressures, are most likely to be beneficial. In cases of provokable outflow tract obstruction, beta-blockers have been shown to be of benefit, and calcium-channel blockers may also prove useful.

## Conclusion

In the 23 years since it was described by Dr. R.D. Teare, hypertrophic cardiomyopathy has been a focus of an ever-increasing amount of research and controversy. This paper has reviewed some of the major areas of active research. While the contribution of genetics has been clarified, the roles of other factors — catecholamines, hyperthyroidism, hypertension, and structural heart lesions — remain to be proven. Most deaths are sudden, and attributed to arrhythmias. While the pathological basis for these arrhythmias has been demonstrated, good criteria for identifying high-risk patients, and regimens for treating and following them, have yet to be established. The crucial question of whether systolic or diastolic dysfunction is the primary abnormality is not yet resolved, and hence the value of surgical versus medical treatment is not yet clear. While many questions and areas of controversy remain, the fact that so much has been learned about a disease first described only 23 years ago, is something of which cardiologists can be proud.□

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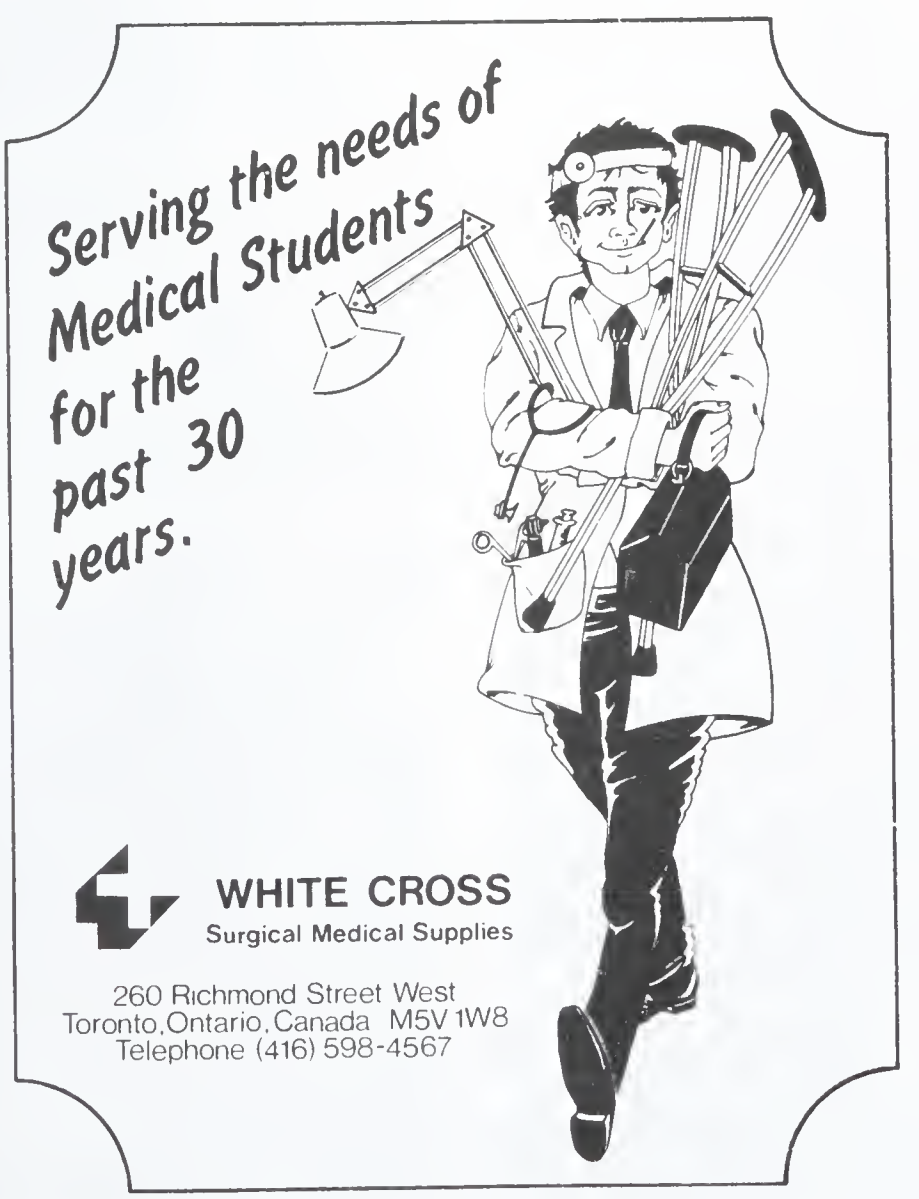

# Breaking Necks In Ankylosing Spondylitis

Tom A. Stiffel

For the past number of years, her view of the world had been going downhill — literally, downhill. It had begun at age 13 with low back pain and stiffness with painful swelling of all her joints. By the time Jewel was 16, she could no longer walk, her hips were fused and she couldn't separate her legs. Then, over the years, her spine began to bend forward. At first, she was embarrassed by the crook in her back. Then, when her neck slowly began arching forward and downward in a rigid curve, she could no longer see people's faces and now, at 42, all she saw were their feet.

This unfortunate lady was suffering from a severe form of a disorder that affects about 1 in 250 people to some degree — *ankylosing spondylitis (AS)* — a chronic inflammatory arthritis of the spine (spondylitis) and several other joints, which ultimately leads to bony fusion (ankylosis) and subsequent deformity. Also known as Marie-Strumpell disease. It was first accurately described by Ernest Strumpell, a German pathologist, in 1884, but it had been recognized long before this. In fact, skeletons with the crippling effects of this disease have been found dating back to the 14th century B.C.[1]

The etiology still remains a mystery, however. There is a marked association with the HLA-B27 antigen: it is found in 97% of patients with AS, in 51% of first-degree relatives, but in only 4-6% of controls. However, only 5% of men and fewer than 1% of women with the HLA-B27 antigen ever develop AS. It appears that possible environmental factors such as genito-urinary or bowel infections act as triggers in genetically - predisposed patients. Males are affected four times more frequently than females.[2] The significant male preponderance of severe cases as well as the increase in AS incidence during puberty and early sexual maturation suggest a possible hormonal factor in the expression of the disease.

The prevalence of AS based on clinical findings is 5.8/1000 in males and 0.8/1000 in females among whites. The highest prevalence of 62/1000 is found in the Haida Indians of British Columbia, 50% of whom are HLA-B27 positive.[3]

## Clinical Features

The classical presentation is that of a young male with slowly progressive low back pain and stiffness over several months. The sacro-iliac joints are usually the first to be involved. Radiographs demonstrate the progression from marginal erosions with poor joint definition, to periarticular sclerosis and ultimately, to bony ankylosis (fusion) and obliteration of the joints. This frequently takes as long as twenty years.

The spine can be affected at any level but the disease usually progresses upward from the lumbar to cervical regions. The vertebrae become squared and bony bridges (syndesmophytes) form within the annulus fibrosis between adjacent vertebral bodies, resulting in the classic "bamboo spine" on radiographs. The normal lordosis of the lumbar spine is lost, as is that of the cervical region. This causes the head to become rigidly bent forward in kyphosis, and is accentuated by the increase in the normal thoracic kyphosis which also occurs. The marked rigidity of the cervical spine also makes it highly susceptible to fracture with the risk of quadriplegia.

Twenty percent of patients with AS present solely with peripheral joint involvement (usually monoarthritis of the knee or ankle). Proximal joint involvement such as in the shoulder or the hip are more likely to progress to permanent damage and limitation of movement. Fixed flexion deformity of the hips is frequently the cause of limited mobility besides the spinal stiffness,and gives rise to the characteristic rigid gait. Thus, prior to the correction of any "spinal" deformity, the degree of fixed hip flexion must be assessed, and indeed may be the most significant cause of the apparent deformity. The patient may also be plagued by plantar fasciitis, Achilles tendonitis and ankylosis of the symphysis pubis and costochondral joints.[4]

There are a variety of interesting extra-articular manifestations. Iritis usually responsive to treatment oc-

curs in 25% of patients. Cardiac involvement occurs late in up to 10% of patients with a 30-year history of AS. The most important cardiac complication is aortic insufficiency due to inflammation of the root of the aorta and aortic valve which results in dyspnea and angina. The thoraco-lumbar kyphosis and fixed chest wall are responsible for a decrease in chest expansion and may result in cor pulmonale. In the late stages, the cauda equina may be affected and this should be suspected when there is insidious onset of urinary or fecal incontinence.

The clinical course is variable and unpredictable. Forty percent have only slowly progressive backache and stiffness with no constitutional symptoms and minimal disability. Fifty-five percent are more severely affected with restriction of back movement and a noticeable thoraco-lumbar stoop by 10 years, often accompanied by peripheral joint involvement. In the remaining five percent, progression is rapid from the onset with severe disability within a few years.

## Treatment

Treatment is aimed at slowing or halting progression of the disease and preventing the crippling spinal deformity. The patient is encouraged to lead as normal a life as possible. Daily breathing and muscle exercises are taught. Swimming is an ideal exercise. The use of a hard upright chair and a hard mattress without pillows is stressed. Physiotherapy has been shown to be an important factor in preventing deformity and improving chest expansion. Phenylbutazone (300 mg daily in divided doses initially, with a maintenance dose of 100 mg thereafter) is anti-inflammatory and also gives symptomatic relief, sometimes striking in degree. Other anti-inflammatory agents (eg. Indomethacin, Naprosyn), although less potent may be used in the 25% of patients who cannot tolerate phenylbutazone. Steroids should only be used for complications of AS such as iritis and plantar fasciitis. Radiotherapy to the spine is no longer used due to the risk of hematologic complications, including malignancy (leukemia).[5]

As mentioned previously, 5% of patients deteriorate rapidly from the onset, while 55% end up with progressive spinal deformity after 10 or so years. Thus, up to 60% of patients with AS have some degree of spinal deformity. Those who are severely disabled by their rigid forward stoop, unable to see the ground more than a few feet ahead- an embarrassing as well as dangerous problem-can now be helped to a remarkable degree by orthopaedic surgery. It was not always so. It took such pioneers as Dr. E. H. Simmons, Director of Orthopaedics at the Toronto East General and Orthopaedic Hospital, to perfect the technique of spinal osteotomy and to apply it to the correction of kyphotic deformities not only of the lumbar and thoracic regions, but also of the cervical area; an extremely hazardous task due to the inherent risks of permanent neurologic impairment.

## "Breaking Necks"

Dr. Simmons first began literally breaking necks over 10 years ago. Previously, in the rare cases where the operation was resorted to, it was done under general anesthesia. More often than not, the patient did not awake from the anesthetic. Under local, on the other hand, the patient could indicate to the surgeon if he developed any symptoms of spinal cord compression when the neck was actually broken, thus preventing any serious sequelae.

At the time, it seemed rather daring to do such a major operation under local anesthesia and the anesthetic service strongly resisted the suggestion at first. It was only after endotracheal intubation was attempted without success in the first of Dr. Simmons' patients, that local anesthesia then became the only feasible method.

Dr. Simmons made his first attempt in 1967. Says he, "The first one was a man from the "Soo". He was a good fellow, French Canadian, bent down, chin on his chest, 33 years old. He hadn't worked for 6 years and everybody in town looked at him as a cripple. I said to him, 'There are certain things you should know about this. It hasn't been done before, and if we do this corrective surgery on your neck, which is a pretty dangerous area, there are two major potential risks. The first and perhaps the least severe is that it could kill you. The second and perhaps the worst is that you could be left paralyzed. I don't think we will have either of these problems, and I wouldn't do the operation if I didn't think it could be successful, but the risks are there.' And he said, 'Look, I'm no good as I am, and if you think there's a reasonable chance, I'd like you to go ahead.' And that's pretty well the philosophy of most of these people."[6]

## Operative Procedure

Preoperatively, the patient is fitted with a plaster body jacket having supports for a halo unit. A halo metal device is fitted to the skull with four screws. The operation is done with the patient in the sitting position with traction applied to the neck through the halo, along the line of the neck. The halo also serves postoperatively to stabilize the head to the body jacket.

The exposure is done from the posterior aspect of the neck under local anesthesia. The goal of the osteotomy is to remove a wedge of bone posteriorly from the C7-T1 level so that by extending the neck, it can be straightened from its previous kyphotic position and fused in a new, more functional position. The C7-T1 level accommodates the correction best, as the spinal canal is relatively wide at this level, the cervical cord and the eighth cervical nerve roots are readily flexible, and any weakness due to eighth nerve compression causes less disability than injury to other roots. Also, the vertebral ar-

teries enter the spine at the transverse processes of C6. This protects them from injury by surgery at the C7-T1 level.

With adequate exposure, the C7 spinous process and laminae are completely removed along with the inferior portion of the arch and spine of C6 and the superior portion of the arch and spine of T1, as are the adjacent parts of the transverse processes of C7 and T1. By removing this wedge, the dura and spinal cord are exposed, and the eight nerve root decompressed. Any irritation of this root can be verbally localized by the patient.

Prior to extending the neck, the patient is given a small amount of anesthetic by mask until he no longer can hold it to his chin. The neck is then gradually extended until the spine fractures anteriorly. Further extension causes the lateral masses posteriorly to come into apposition, while a central gap remains open to prevent compression of the cord due to buckling of the dura. After routine closure, the head is stabilized with the halo apparatus. The patient is fully conscious and is asked to move all four limbs to assess his neurological status. He is able to stand and walk to his waiting bed.

Post-operatively, the patient is nursed in a circo-electric bed, which moves the supine patient from the horizontal position through 90 degrees to the vertical and allows early ambulation. The patient is discharged home to convalesce with the halo usually being removed at 4 months depending on the degree of union radiographically. It is replaced by a cervical brace support for a further 2 months. It is important that the angle of correction be such that a compromise is made between allowing the patient to look ahead for walking, and being able to read and write at a desk, as the neck and entire spine remain completely fused.[7]

## A Success

The results of this technique in Dr. Simmons' hands have been most remarkable. Of the 42 patients operated on between Oct. 1967 and Aug. 1976, non-union occurred in only 2 patients. These were successfully treated with anterior cervical fusion at the C7-T1 level using the Keystone method. In only one patient was further decompression of the eighth nerve root required. There were three other post-op complications but these were unrelated to the cervical osteotomy itself.[8]

Thus, a procedure that previously left the patient dead more often than not, has become under Dr. Simmons a safe and reliable method to correct severe kyphotic deformities of the cervical spine in AS.

Back on ward rounds, Dr. Simmons, the neck breaker, speeds into another room. " 'Hi, Jewel, honey, how are you? Are you feeling better? Are you sore there?' Simmons says, feeling the muscles in her neck. 'How's it feel looking ahead? Can you see the ceiling?' he asks. 'I felt funny at first, I liked it!' Jewel says, shyly smiling."[9]

It's difficult to describe the difference between living as a normal person and as a cripple, but the expression of gratitude and hope on her face said it all. Jewel can now look ahead and see her children for the first time in years. At her home in Colorado, she is presently active in social work helping others with this crippling disease.[10] □

*Tom Stiffel is a graduate of the McGill University medical school.*

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# Applied Surgical Anatomy Of The Facial Nerve

Jerry B. Naiberg 8T3

*A detailed understanding of the anatomy of the facial nerve is required by all those who manage patients with facial paralysis. Furthermore, an intimate knowledge of the course of the nerve through the temporal bone is mandatory if otological surgery is to be safely and successfully performed. A number of specific investigations have been devised to assess the functional integrity of the various branches arising within the bone. With these tests it is now possible in many cases to determine the precise location of a lesion affecting the facial nerve. The purpose of this paper is to review the anatomy of the facial nerve and discuss the methods now available for localization of a lesion affecting the nerve.*

## Anatomy

The facial nerve is commonly referred to as the nerve that supplies the muscles of facial expression. While this definition is correct it is by no means complete; it is hardly possible to alter facial expression by contracting the stapedius muscle!

A study of the embryology reveals that the seventh cranial nerve supplies the muscles derived from the motor plate of the second branchial arch, and this description is preferred. These muscles include the stapedius muscle, posterior belly of digastric muscle and the stylohyoid muscle, together with the muscles of facial expression (buccinator, auricularis, frontalis, platysma, orbicularis oris and oculi).[1]

The cell bodies of the motor portion of the nerve are found in the motor nucleus situated in the ventrolateral part of the pontine tegmentum.[2] Other fibers run with the facial nerve for at least part of their course. These are: 1) parasympathetic secretor-motor fibres running from the superior salivatory nucleus to the sublingual and submandibular glands via the chorda tympani and similar fibres running from the lacrimal nucleus to the lacrimal gland via the greater superficial petrosal nerve; 2) afferent taste fibres from the anterior two thirds of the tongue which reach the gustatory nucleus via a peripatetic route, part of which includes the chorda tympani and the facial nerve; 3) there is indirect evidence of a sensory branch of the facial nerve which innervates a small portion of the external ear. This consists of the appearance of vesicles on the external ear in cases of herpes zoster affecting the geniculate ganglion.[3]

The facial nerve enters the temporal bone through the internal auditory meatus anterior to the superior vestibular nerve and above the chochlear nerve. It travels laterally in the bone until it reaches the geniculate ganglion. At this point the greater superficial petrosal nerve arises and proceeds anteriorly (fig. 1) while the rest of the facial nerve makes an approximately 90 degree turn and continues posteriorly on the bony facial (fallopian) canal. This canal makes up part of the medial wall of the middle ear and is situated above the oval window (and stapes) and below the lateral semicircular canal. The nerve continues posteriorly until it reaches the mastoid region of the temporal bone. At this point the nerve makes another 90 degree turn and travels inferiorly until it reaches the stylomastoid foramen.

However, in the nerve's final descent through the temporal bone two important branches arise; firstly, the stapedial nerve, innervating the stapedius muscle and secondly the chorda tympani. This latter branch passes forward between the incus and the malleus and leaves the ear through a foramen in the anterior wall of the tympanic cavity (fig. 2).

As the main trunk of the facial nerve emerges from the stylomastoid foramen it soon enters the body of the parotid gland, surgically dividing the gland into

deep and superficial lobes. It then usually divides into 2 divisions and then these 2 divisions further subdivide into 5 main branches. These 5 branches have been likened to a goose's foot (*pes anserinus*). Unfortunately for the surgeon performing operations on the parotid gland the pattern of branching is variable (fig. 3)[4]. It is only through a painstaking dissection of the nerve within the gland that the possibility of a traumatic facial paralysis is avoided.

## Facial Paralysis

The first step in the management of a patient with facial paralysis is to determine if the lesion is upper motor neuron or lower motor neuron in type. Characteristically, upper motor neuron lesions cause paralysis of the lower two thirds of the face, sparing the forehead, while lower motor neurons lesions usually involve the entire hemi-face. The treatment and prognosis for a patient with a lower motor neuron facial paralysis will depend on the cause of the lesion and its location. There is a spectrum of nerve damage ranging from axonal degeneration distal to the site of the lesion which carries a poor prognosis to a physiological block (neuropraxia) in which there is no axonal degeration and in which complete recovery can be expected. The type of lesion can be determined using a nerve excitability test. This test consists of placing an electrode over the stylomastoid foramen and stimulating the nerve percutaneously with a square wave pulse. The minimum current required to cause a twitch in the facial muscles is recorded on the normal side, and the affected side is then tested. If the nerve is neuropraxic, a muscle twitch will be elicited with the same level of current. If the nerve has partially degenerated, more current will be required to elicit the twitch. If the nerve is completely degenerated, a maximal stimulus will fail to elicit a response. This test has the disadvantage that axons that have been severed can still be stimulated distal to the lesion for up to 72 hours. Therefore nerve excitability tests can only be used 3 days after the onset of paralysis. Nevertheless this test is simple to carry out and continues to have a definite place in clinical practice. [5,6,7]

Evoked electromyography is a useful technique for evaluating cases of facial palsy. This test is similar to nerve excitability testing but instead of evaluating the function of the nerve on the basis of an observed muscle twitch, electrodes are used to detect muscle action potentials. This technique allows a quantitative evaluation of nerve excitability, conduction velocity and amplitude and duration responses to be made and is more sensitive. [6,7]

Electromyography can also be used for the detection of fibrillation potentials. These myogenic potentials are characteristic of muscle fibres that have been denervated for more than 10 days. Absence of fibrillation potentials in early cases of facial paralysis (2-10 weeks) indicates that denervation has not occurred and that a good functional recovery can be expected.[3]

If fibrillation potentials are observed, a prolonged recovery with the probability of eventual impaired facial function is likely. Gradual fading of fibrillation potentials after 10-12 weeks indicate that reinnervation has begun and that recovery should be at least partial. Gradual fading of fibrillation potentials after 1 year indicates that the muscle involved has atrophied and that no recovery can be expected.[3]

The etiology of lower motor neuron facial paralysis can be quite varied. Although most cases of facial paralysis are still classified as idiopathic, some of the known common causes include: herpes zoster cephalicus, surgical trauma, accidental trauma, complications of otitis media, cholesteatoma and neoplasms of the nerve itself and the parotid gland. [8,9]

In cases of idiopathic facial paralysis *(Bell's Palsy)* it is important to localize the site of the lesion. Firstly, the location of the lesion may determine if surgical decompression of the nerve is to be entertained. Secondly, studies of this type may delineate the etiology of this disorder.[10]

Lesions in the facial nerve resulting in facial paralysis can be localized to four regions: 1) proximal to the geniculate ganglion, 2) between the geniculate ganglion and the stapedial nerve, 3) between the stapedial nerve and the chorda tympani, 4) distal to the chorda tympani. The following tests are used to determine the precise site of the lesion.

## Schirmer's Test of Lacrimation

Function of the greater superficial petrosal nerve and its central connections can be assessed using this test. Absorbant strips of filter paper are hooked into the lower conjunctival sac of each eye simultaneously. If the amount of fluid absorbed on the affected side is significantly less than the amount absorbed on the normal side, then this pathway is not functioning normally. The facial nerve lesion, then, is proximal to the geniculate ganglion.[11]

## Stapedial Reflex Test

In the normal ear the stapedius muscle contracts in response to loud sound. This causes the head of the stapes to be pulled away from the oval window and the osicular chain becomes less compliant i.e. its impedance is increased. This increase in impedance can be detected by using tympanometry. In this test a probe is placed into the ear on the affected side. This probe conducts sound from a source generator into the ear canal and also measures the amount of sound reflected from the outer surface of the tympanic membrane. If a loud sound (80-90dB) is introduced into the other ear, a stapedial reflex will be elicited in both ears (the reflex is bilateral). This will cause the ossicular chain to stiffen which in turn will cause the tympanic membrane to stiffen and thereby increase its

impedance. This will cause more sound to be reflected from the tympanic membrane and this change will be detected by the apparatus. If no stapedial reflex can be elicited, the facial nerve lesion is proximal to the stapidial nerve. This test, of course, requires that hearing is normal or near normal on the unaffected side.[12]

## Electrogustometry

This test evaluates the integrity of the chorda tympani. The basis for this test is that a small electrical current applied to the tongue will evoke the sensation of a metallic or bitter taste. In patients with facial paralysis, an increase in electrical current is required to elicit a taste sensation on the affected side, as compared to the normal side, is evidence that the facial nerve lesion is proximal to the chorda tympani.[13]

Table 1 gives a summary of the results obtained for the various tests for facial nerve lesions at various sites.

## Management

Treatment of patients with idiopathic facial paralysis is very difficult. The major problem is that as many as 70% of these patients will have a spontaneous recovery and both medical and surgical methods of management carry risks of their own.[14]

Presently there is a great deal of controversy with regard to the treatment of idiopathic facial nerve paralysis. One view is that this type of lesion is due to ischemic compression of the facial nerve within the temporal bone and that surgical decompression is the most suitable treatment.[10] Others maintain that this type of lesion is due to reactivation of herpes simplex virus and that the best method of treatment is the administration of large doses of prednisone.[15]

Unfortunately, neither method of treatment has undergone complete clinical trials and further studies in this difficult area are badly needed.[16] Nevertheless, it is mandatory that the management of any case begins with an understanding of the applied anatomy of the nerve and a knowledge of special tests now available which assist the physician in determining the location of a lesion. The question of any unnecessary or potentially hazardous therapy, be it medical eg. high doses of steroids, or surgical eg. facial nerve decompression remain to be resolved. □

TABLE 1

| Location of Lesion | Schirmer's | Stapedial Reflex | E.G. |
|---|---|---|---|
| nuclear | + | - | + |
| proximal to geniculate ganglion | - | - | - |
| in the area of the geniculate ganglion | - | - | - |
| proximal to the site of origin of the stapedial nerve | + | - | - |
| distal to the site of origin of the stapedial nerve | + | + | - |
| distal to the site of origin of the chorda tympani | + | + | + |

+ indicates normal response
- indicates decreased or absent response

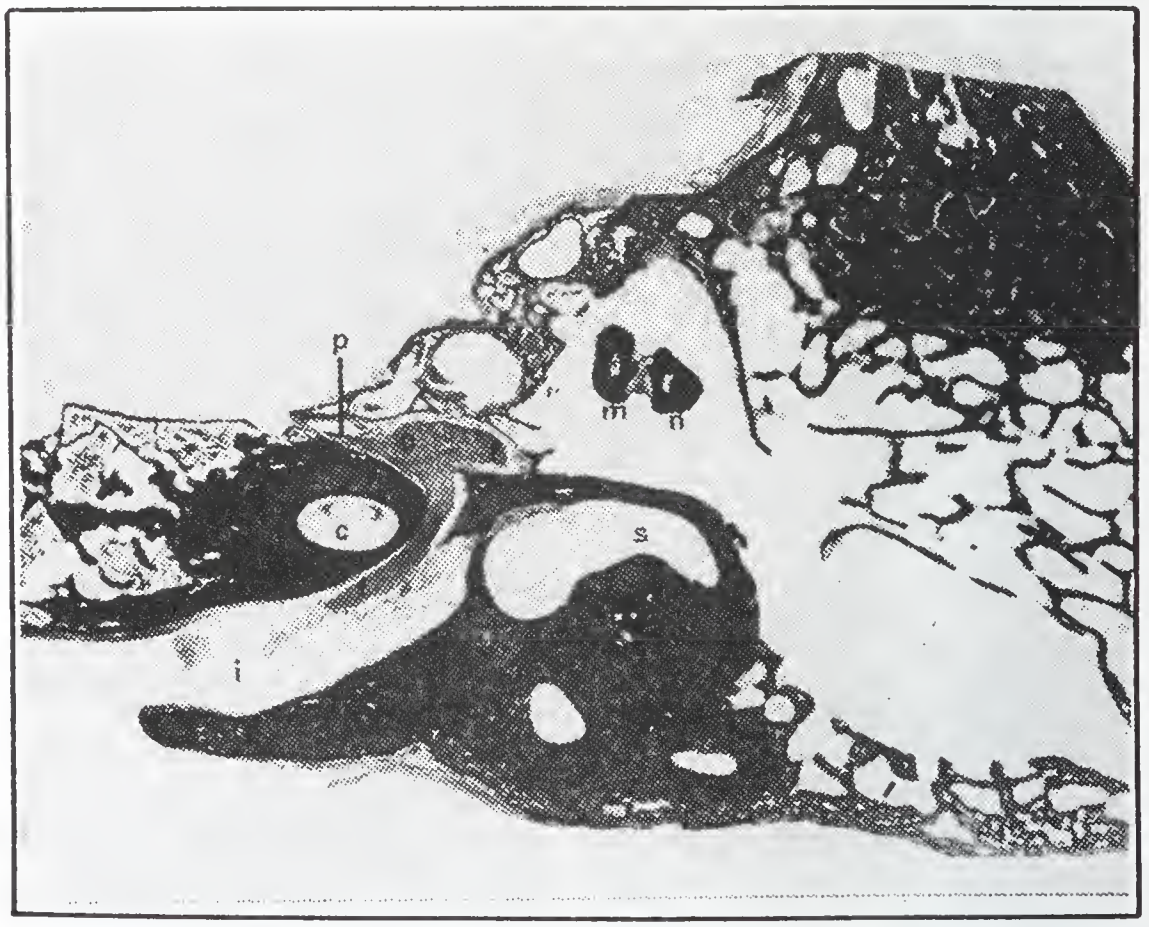


FIGURE 1: Horizontal section of the temporal bone at the level of the genigulate sanglion.

c = choclea,
f = facial nerve,
g = geniculate ganglion
i = internal auditory meatus
m = malleus
n = incus
p = greater superficial petrosal nerve
s = lateral semicircular canal

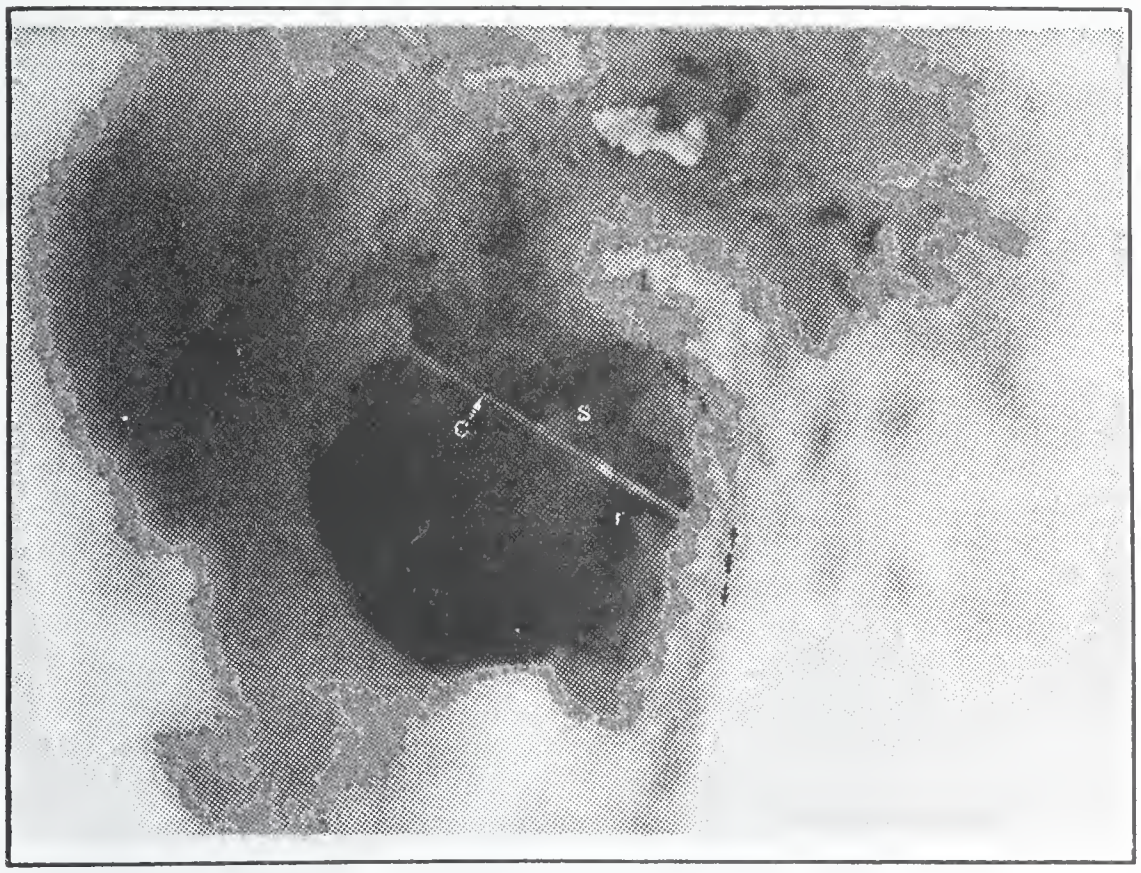


FIGURE 2: Gross anatomy of the middle ear. The posterior wall of the external auditory meatus and part of the mastoid region of the temporal bone as well as the malleus and incus have been removed.
c = chorda tympani
f = facial nerve
l = lateral semicircular canal
r = round window
s = stapes

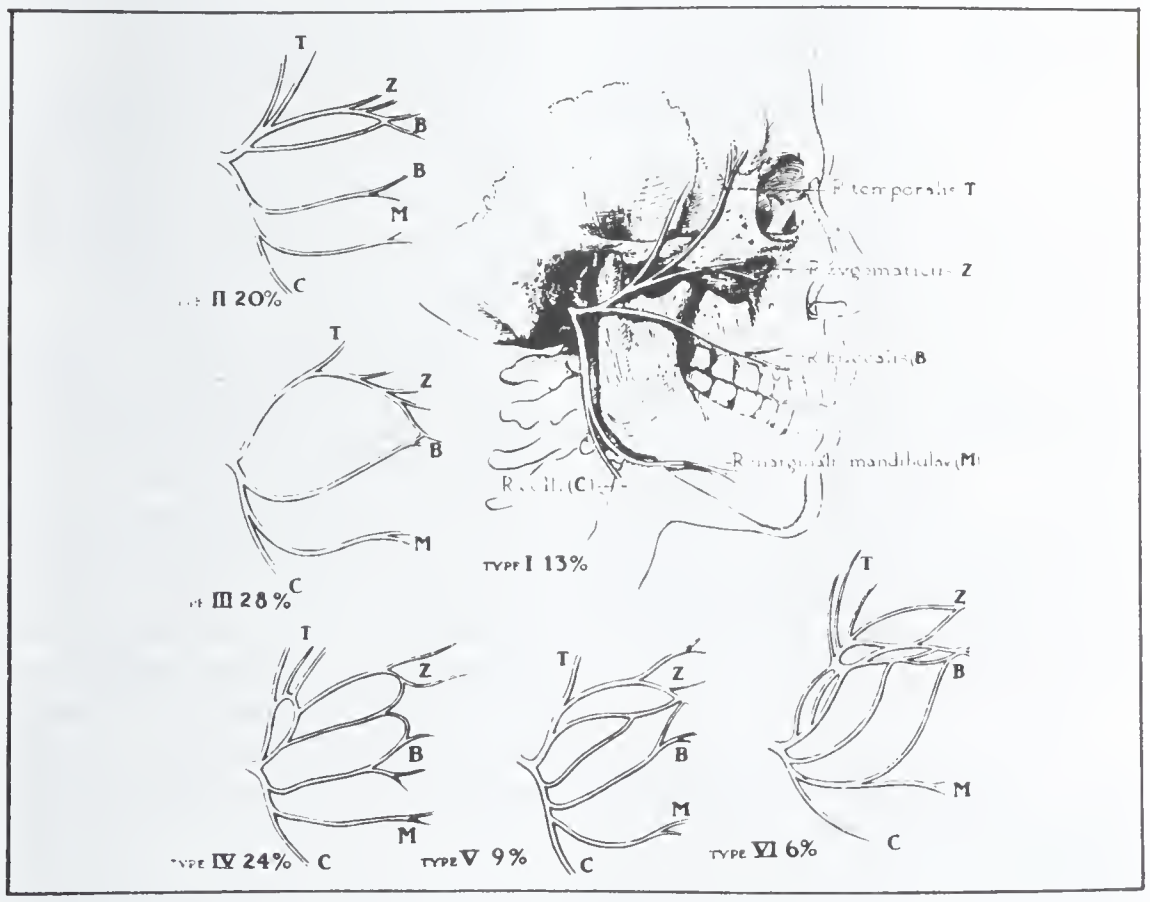


FIGURE 3: Various patterns of branching and their frequency of the facial nerve within the parotid gland. *(Reprinted by permission of Surgery, Gynecology, and Obstetrics).*

## Acknowledgement

This work was carried out during an elective in the Temporal Bone Laboratory, (Director Dr. M. Hawke). The author is grateful to Mr. Malcolm Keene, M.B., F.R.C.S., and Dr. J. Farkashidy for their encouragement and guidance. The author is also grateful to Allison MacKay for photography.

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# Gastroesophageal Reflux In Children

**Michael Dan 8T4**

*The topic of gastroesophageal reflux in children has been reviewed, with special emphasis on the etiology, and history of surgical treatment. As well, pathology and pathophysiology, clinical presentation, diagnosis, and medical/surgical management have been discussed.*

Gastroesophageal reflux (GER) — the regurgitation of gastric contents into the esophagus — although frequently observed in adults, was at one time thought to be quite rare among children. Today it has been shown to be quite common in childhood, especially among infants.

At birth, the physiological sphincter preventing reflux is not fully mature. The lower esophageal sphincter (LES) becomes fully operational only by 5-7 weeks[3]; therefore reflux, regurgitation, and vomiting are often observed even in the normal newborn. In a small percentage of the population, however, the reflux is pathological, and the child eventually shows signs of failutre to thrive, gastrointestinal bleeding, or aspiration pneumonia. Radiological examination of these individuals may show a small herniation of the stomach through the esophageal hiatus, but frequently GER is the only abnormality.

## Historical Background

For many years, physicians evaluating a child with frequent vomiting and regurgitation placed a great deal of emphasis on the presence of hiatal hernia, while failing to recognize the more basic physiologic problem of GER. In 1836 Bright[4] gave the first account of hiatal hernia, discovered at autopsy in an 18 year old girl. It was not, however, until 1947 that GER, or "chalasia", as it was then called, came to be known clinically[15]. This chronological precedence of anatomical description over physiological understanding is interesting in its own right, and has had a profound effect on the philosophy of surgical treatment of GER.

In 1951, Allison[1] proposed a method for repairing hiatal hernia which, although anatomically faithful in its reconstruction of the correct gastro-phreno-esophageal relationships, was only partially successful in eliminating reflux. As late as 1969, the superfluous distinction between GER and hiatal hernia persisted, with each appering as separate entries in Swenson's Textbook of Pediatric Surgery[22].

In 1961, Nissen[16] proposed the first "physiological" surgical procedure to correct GER. His "fundoplication" consisted of creating an effective anti-reflux cuff out of gastric fundus and esophagus. Today it is considered by many to be the surgical treatment of choice. It is described in more detail later.

## Pathology and Pathophysiology

The primary pathology of GER consists of incompetence of the LES. Secondary changes are related to esophagitis, the most serious of which is stricture formation due to scarring. When aspiration is present, pathological changes can extend into the bronchi and lungs.

Anatomical considerations include an almost non-existent abdominal esophagus and an obtuse esophogastric angle (angle of His), both distinguishing features of the immature esophogastric junction. As well, the sphincterlike mechanism at the cardia is absent due to defects in any of the following (1) the phrenoesophageal ligament; (2) the mucosal choke at the esophogastric junction; (3) the diaphragmatic crura. Despite these morphological findings, no physical basis for the LES has yet been established in man[19].

At rest, normal intraesophageal pressure is comparable to intrathoracic pressure, and varies dynamically with inspiration and expiration. Manometric studies of the lower esophagus, however, have established a zone of high pressure (15-25 mmHg) directly above the esophogastric junction. This forms the physiological basis for the LES. Unfortunately, absence of this high pressure zone is not pathognomonic in every case of GER[23]. Additional factors in the mechanism of the LES have yet to be elucidated.

## Clinical Presentation

Clinically, two separate populations exist: infants, and older children including adults (discussion of the latter will be restricted to a few examples only). In the infant, the clinical findings may be broadly grouped into four areas (1) vomiting; (2) esophagitis; (3) aspiration; (4) malnutrition.

Commonest of all signs is *vomiting*, which has been noted to be present in 90 per cent of all cases[17]. The nature of the vomiting is, however, of little diagnostic value — containing flecks of "coffee ground" blood clots in some individuals, and being projectile in others. Occasionally, bile-stained vomitus is observed. Most vomiting occurs after feeding, when the infant is in the recumbent position.

Chronic *esophagitis* leading to mild dysphagia may be present in all affected individuals[14]. Prolonged acid insult eventually leads to diffuse esophageal spasm and stricture formation. Clinically, an aberrant island of gastric mucosa may mimic reflux esophagitis, and ought to be entered into the differential diagnosis. If ulceration is present, bleeding into the GI tract may be sufficient to cause a mild iron-deficiency anemia.

Problems due to *aspiration* affect approximately 20 per cent of patients with GER[14]. The consequences range from chronic pneumonia to prolonged apneic spells. GER has therefore been implicated in the etiology of the Sudden Infant Death Syndrome.

Finally, *failure to thrive* due to *malnutrition* ranks as one of the outstanding features of GER in children[14,17]. If vomiting is excessive, normal growth and development landmarks are not attained.

## Diagnosis

Diagnosis of GER can be difficult since it relies so heavily on the correct appreciation of a whole constellation of clinical findings. In the infant presenting acutely, the signs may mimic any of the following: pneumonia, asthma, vascular ring, H-type tracheoesophageal fistula, pyloric stenosis, or congenital heart disease. An infant with less acute signs may be misdiagnosed as having anemia, hypothyroidism, or lead poisoning.

Traditionally, a roentgenographic diagnosis was considered to be definitive. Routine barium swallow is sometimes the only test necessary. Accuracy can be increased by giving a volume of barium equal to a normal feed, and with the patient in the Trendelenberg position, applying pressure on the abdomen.

Recently[13], the technique of continuous 24-hour esophageal pH monitoring has gained much favour, proving to be more accurate in the diagnosis of GER than the usual radiological methods. Two important parameters are reflux time at a pH $<4$, and time required for a return to a normal pH of 6, or clearance time. In the case of the former, a single episode of reflux lasting more than 5 minutes is diagnostic in both children and adults. A clearance time of 13 minutes is also diagnostic.

Aspiration secondary to reflux can be diagnosed by giving a feed containing $^{99m}Tc$ and later scanning the lung fields. Manometric studies, formerly considered to be of value, are no longer used routinely because many patients with normal LES pressures were still found to reflux[23].

Finally, esophagoscopy under general anaesthesia is of value in confirming the diagnosis of esophagitis, and in assessing the patient as a surgical candidate.

## Treatment

Because of the gradual improvement in LES function as infancy progresses, spontaneous resolution is likely to occur in the majority of individuals not yet demonstrating LES competence by 7 weeks of age. This forms the basis for the medical management of GER. In infants as in adults, assumption of the upright position ($>60^0$) at all times is usually curative. In addition, feedings may be thickened by the addition of cereal, and their volumes reduced and frequency increased. Antacids, although of proven benefit in adults, are of little help to infants. At present, no anti-cholinergic or anti-spasmodic medication has been of value — medical therapy is successful in 85 per cent of all infants. Typically, the response is within the first week or two of treatment.

In the 15 per cent of cases that demonstrate persistent vomiting, poor weight gain, or failure to respond to medical therapy, surgical management is indicated. Surgery is also required for individuals with (1) recurrent pneumonia; (2) recurrent GI bleeding; (3) substantial hiatal hernia; (4) stricture formation.

Twenty years ago the classical procedure involved reduction of the hiatal hernia and reconstruction of the correct anatomical relationships. To this a vagotomy and pyloroplasty were often added[6], because of the belief that infants with GER refluxed hyperacidic gastric secretions and exhibited delayed gastric emptying. Only the latter claim has been substantiated[11]. Gradual realization that the real problem is reflux, and not hiatal hernia, has led to the development of effective anti-reflux surgery..

In order to be successful, an operation must (1) lengthen the intra-abdominal esophagus, and (2) create an effective anti-reflux valve. Of widest acceptance are the Nissen[16] fundoplication, Belsey[21] Mark IV fundoplication, and Hill[9] posterior gastropexy. These procedures are very safe, and are effective in over 90 per cent of all patients.

Delay in surgical intervention may ultimately lead to secondary stricture formation. This may be left alone at operation, and resolve itself spontaneously, or may be overcome with a series of dilatations with mercury bougies. If

this fails, primary resection, or Collis gastropexy in combination with one of the above fundoplication procedures may be necessary.

Normally, the most frequent complication of anti-reflux surgery is the "gas bloat syndrome", but this usually disappears within 3 weeks post-operatively.

## Etiology

Because of the many questions still surrounding GER, discussion of the etiology has been put off until last. Before this can be done, however, some additional physiological and pathophysiological findings must be brought to light.

In the past, clinicians tended to over-emphasize the importance of hiatal hernia. Bettex[2], in a recent review of 112 cases of fundoplication for GER and hiatal hernia, described sliding hiatal hernia as only one element in a broad spectrum of disorders ranging from "chalasia" to "upside-down stomach".

Although no pharmacotherapy is presently recommended for GER in children, the observation has been made that gastrin[8] significantly increases LES tone in the digestive state in the opossum. Studies of adult reflux patients[5] revealed a substantially reduced increase in LES pressure to pentagastrin, but no significant difference in basal gastrin secretion, suggesting additional factors in the generation of LES pressure. Basal secretion of the other gut hormones appears to be normal[14].

Recently, motilin[10] has been implicated in the generation of LES pressure in the interdigestive state in the dog. Its actions are abolished by feeding or by infusion of pentagastrin. It appears to play a major role in the control of LES pressure.

Finally, pyloric stenosis accompanying GER, although often overlooked in some of the smaller reviews[7,12], has been consistently reported by others[6,11] to be around 10-15 per cent.

The etiology of GER in children, therefore, involves a consideration of (1) embryological factors; (2) physiological factors; (3) biochemical factors.

In 1952, Roviralta[20] coined the term "phrenico-pyloric syndrome", and suggested that pyloric obstruction might form the basis for GER. A recent report by Redman[18] described 3 cases of hypertrophic pyloric stenosis developing within days after repair of a congenital diaphragmatic hernia. Since the foramen of Bochdalek and the esophageal hiatus are embryologically related to the developing diaphragm, the etiology of GER may be rooted in some type of congenital malformation involving diaphragm and possibly pylorus.

Alternatively, GER could be the result of a neuromuscular lesion, as originally suggested by Neuhauser and Berenberg[15] in 1947. Taking this one step further, it would be reasonable to propose a neurotrophic imbalance between esophageal hiatus and pylorus, with atrophy of the former and hypertrophy of the latter.

Finally, with the recent implication of motilin in the physiology of the LES, it would be equally reasonable to propose a biochemical lesion as the basis for GER. Since circulating levels are no different in reflux patients than in controls[14], one might hypothesize a deficiency of motilin receptors on the smooth muscle that surrounds the distal third of the esophagus in the infant with GER.

At present, management of GER in children seems to be quite satisfactory. This is mainly due to the steady increase in physiological knowledge over the past 30 years. Despite this, more work still needs to be done at the basic and clinical levels in order to reduce the need for surgical intervention and the inconvenience it imposes.□


## Acknowledgements:

The author would like to thank Dr. Robert Filler, Surgeon In-Chief for The Hospital For Sick Children, as well as the other members of the Department of Surgery, Drs. S. Ein, B. Shandling, J.S. Simpson, and C.A. Stephens, for their assistance in the preparation of the manuscript.


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# Hypophysectomy

Walter P. Vanhelder, M. Eng., Ph.D. 8T4

## The use of Hypophysectomy

Hypophysectomy is a basic surgical operation which is used to improve a number of pathological conditions. These conditions can be as follows: metastatic carcinoma of the breast and, prostate, some cases of juvenile diabetes mellitus, diabetic retinopathy, acromegaly, pituitary hyperthyroidism, and Cushing's syndrome [1,2,3].

## The Technique of Hypophysectomy

Various techniques of hypophysectomy can be used [3,4,5]. Some of them employ the actual removal of the hypophysis, or sectioning of the pituitary stalk. Extirpative hypophysectomy can be done either transcranially, or transethmoidally. Transsphenoidal hypophyseal stalk sectioning may involve either barrier implanation, or partial hypophysectomy. Other methods of destroying the hypophysis include radiation implantation, transcutaneous irradiation, radiofrequency coagulation, and cryosurgery.

The technique of hypophysectomy is very important. Besides the successful destruction of the pituitary and/or its function, the degree of inflicted trauma and the cosmetic damage to the patient are other factors which should be considered. Hypophysectomy has generally very low mortality and morbidity.

Two of the most important techniques of hypophysectomy are the transcranial and transphenoidal methods. The latter method has some definite advantages—it is not too traumatic, and it does not leave a visible scar. This method, as used by Cushing[4], was later abandoned by him because risk of an infection was high before antibiotics were available. Today, however, with the use of proper antibiotics the method is quite safe.

Besides its use as a therapeutic tool in human patients, hypophysectomy is a surgical technique used in experimental animals. Hypophysectomy in a dog is necessary for some basic investigations in the field of endocrinology. One of the important methods, if not the most suitable of all, is the interbuccal approach of hypophysectomy in the dog. In this method the surgeon approaches the pituitary gland through the soft palate and the sphenoid bone of the animal. This unique operation was improved by H. Essex and T. Astarabadi[6] in 1953, and by J. Markowitz and J. Archibald[7] in 1955.

## The Interbuccal Method of Hypophysectomy In The Dog.

The previously anesthetized animal is placed on its back on the operating table. The control of salivation is achieved by administration of atropine. The mouth of the dog is opened and the upper jaw is fixed by a string to the table (Fig. 1). The lower jaw is attached to a special stand by pulling the tongue over a horizontal rubber covered bar and by fixing it in that position with adhesive tape. The hard and soft palates are sponged with Zephiran solution.

The primary landmarks of the incision are the hamulate processes of the pterygoid bones (Fig. 2). Their exact location is established by palpation. A 3cm incision is made by a scalpel, perpendicular to the line connecting both hamulate processes. A Weitlaner retractor is used to pull the tissues of the soft palate aside (Fig. 1). As the bleeding starts, a suction pump is employed, and is used intermittently to dispose of the blood. The mucoperiosteum is removed to the sides by a periosteal elevator. Thereafter, in most cases, two other landmarks—the intrasphenoid suture, and the emissary vein — become visible (Fig. 2). A dental drill, approx. 4mm in diameter, is aimed caudad to the emissary vein. An important anatomical feature must first be realized: the circular sinus completely surrounding the pituitary in the dog. The sphenoid consists of three layers of bone — harder outer cortex, softer spongy middle layer, and harder inner cortex. These differences are reflected by the resistance which the bone gives to drilling. This must be observed carefully by the surgeon. When the drill reaches the inner

hard layer it is necessary to proceed with caution in order not to accidentally thrust the drill through the sphenoid, destroying the pituitary or the surrounding and underlying structures. Bone bleeding is controlled during drilling by putting small marble-like pieces of bone wax into the opening in the sphenoid. The drill presses the wax into the walls of the opening and also removes any excessive wax. When the rest of the inner bone layer is cautiously removed by the drill, the pituitary is seen bulging under the dura mater. Using a curved scalpel, an incision is made in the dura mater overlying the hypophysis. cerebrospinal fluid can usually be seen seeping out of this incision. The suction pump is applied against the pituitary bulging through the incision in dura mater. The suction which is increased up to 25 atmospheres, detaches the gland from its stalk.

Thereafter, the opening in the sphenoid is plugged by bone wax and the mucoperiosteum replaced to its original place. The soft palate is sewn together using interrupted stitches. Any blood which has accumulated in the nose of the animal is removed by the suction pump. An injection of a suitable antibiotic is administered to the animal, and the proper postoperative care is started.

This author believes that the described method of hypophysectomy can be of interest to medical students interested either in surgery or in research.□

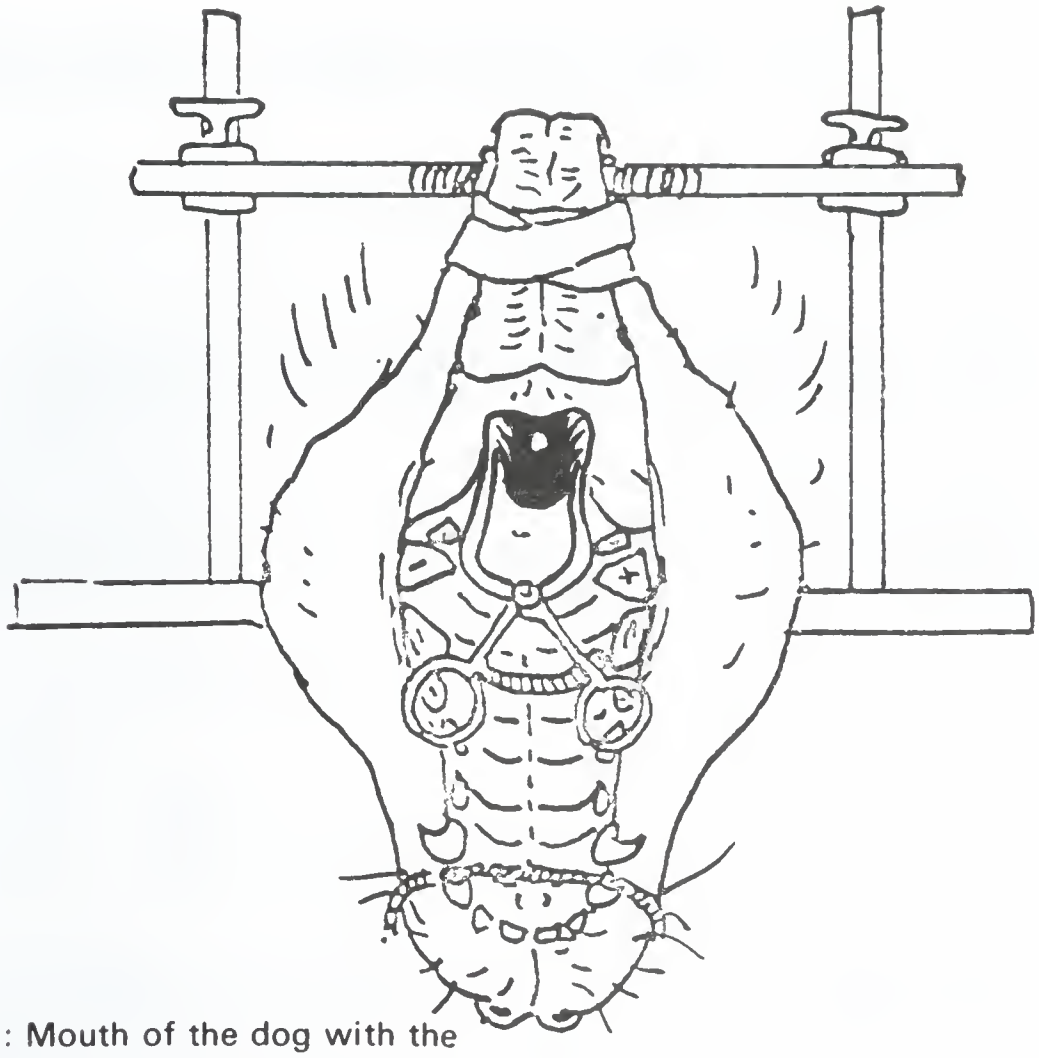

Fig. 1: Mouth of the dog with the Weitlaner retractor inserted into the incision in the soft palate.

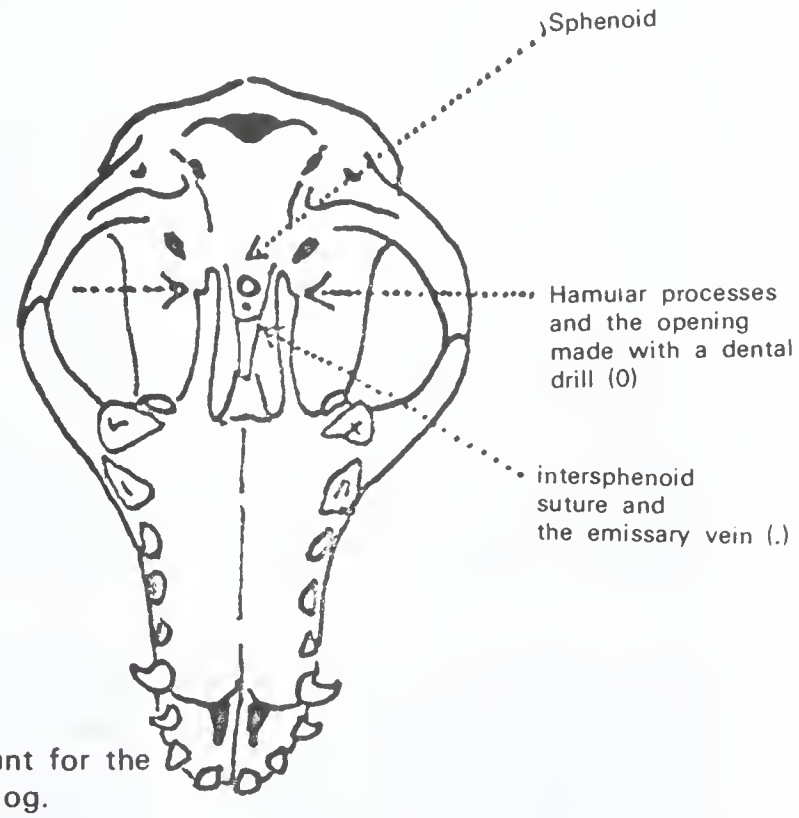


Fig. 2: Landmarks important for the hypophysectomy in the dog.

**Acknowledgements:**

The author of this article learned this technique from Dr. A. Sirek, a former associate of J. Markowitz and J. Archibald.

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# Streptase (Streptokinase):

## A Significant Advance In Resolving Thrombosis and Embolism.

Although a blood clot can prevent someone from bleeding to death during injury, it can also create serious illness and death in other circumstances. One such situation is deep vein thrombosis, a condition in which blood clots adhere to inflamed portions of a vein. Its incidence is high among postoperative patients with prolonged bed rest as well as postpartum patients.

Uncomplicated deep vein thrombosis is quite benign. Major complications include the least severe *popstphlebitic syndrome* and the potentially lethal *pulmonary embolus*. The former presents as ulcers and swelling of the ankles, due to permanently damaged valves of the deep leg veins. The latter occurs when a free thrombus from the leg becomes lodged in the pulmonary circulation. The size of the embolus as well as the vessel plugged will determine the severity which includes sudden death.

The traditional therapy for deep vein thrombosis and pulmonary embolism is complete bed rest and anticoagulation. The latter is started by intravenous heparin, which is replaced after a few days by a second anticoagulant from the coumarin group of drugs. Although anticoagulation is effective in many cases of deep vein thrombosis and pulmonary embolism, it is far from being perfect. First of all, it does not dissolve the existing thrombi which may embolize. Secondly, it does not return the damaged lung circulation to normal. Thirdly, damage to the valves of leg veins is not prevented, hence, the postphlebitic syndrome occurs. Therefore, anticoagulants only allow the body to dissolve existing thrombi by its natural mechanisms over an extended period of time. Since seriouly ill patients with large clots cannot afford that amount of time, their only option is surgical removal of the offending clots even through surgery is extremely risky in these patients.

Only July 1, 1981 Streptase (streptokinase), a protein derived from group C-beta-hemolytic streptococci bacteria, was released for general distribution in Canada. Streptase converts naturally — occurring plasminogen in a patient's blood to the proteolytic enzyme plasmin. Plasmin then works in dissolving clots. By switching on the body's clot dissolving system, Streptase is able to resolve thrombi and emboli and thus restore the circulation to normal more readily. Moreover, mortality may be reduced by normalizing the circulation. Also it helps to prevent permanent damage to the valves of the veins and to the pulmonary vasculature. Streptase, in combination with anticoagulants, decreases recovery time and the necessity for surgery in many cases.

Nevertheless, the most common problem associated with Streptase and anticoagulants is unwanted bleeding caused by the indiscriminate action of active plasmin on pathological clots as well as therapeutic hemostatic plugs. Fortunately, bleeding from resolving hemostatic plugs in quite minor usually. Even a major bleed can be controlled readily by transfusion with whole blood or blood products. Actually, the incidence of unwanted bleeding in these patients approximates that in patients treated with anticoagulants alone.

Apart from treating deep vein thrombosis and pulmonary embolus, Streptase is also indicated to help in resolving arterial thrombi and emboli, and for clearing plugged arteriovenous cannulae. □

(The author would like to thank the Kerbel Communications Group for sending the above information).

*James Chu 8T3*

# Recent Innovation In Analgesic Therapy

Since currently available analgesics, either opiates or non-opiates have certain drawbacks. the introduction of a new, orally effective and well-tolerate non-addicting, non-narcotic analgesics is certainly an important therapeutic advance. Zomepirac Sodium (Zomax®) is such an advance.

Zomepirac sodium was discovered in 1969 by McNeil Pharmaceutical and first tested in clinical trials in 1975. Numerous single dose and long term trials have demonstrated that zomepirac sodium to be the first single-entity, oral analgesics which has no addiction potential but is as effective in the treatment of mild to moderately severe pain as the usual does of standard narcotics and non-narcotics (eq. Aspirin). It has also been studied and proven effective in treating many typical kinds of pain, chronic muscle and joint pain, dental pain, sprains and fractures and acute pain following surgery. The efficacy of zomepirac was further confirmed in cancer patients with moderate to severe postoperative pain — oral doses of this drug produced an analgesic effect equivalent to intramuscular doses of morphine.

The mechanism of analgesic action of zomepirac is not exactly known; however, it possess some of the same pharmacologic properties as aspirin and other non-steroidal anti-inflammatory drugs: analgesic, antipyretic, and anti-inflammatory activities. It inhibits prostaglandin synthetase and the aggregation of platelets induced by collagen or epinephrine. Inhibition of prostaglandin synthetase has been postulated to result in analgesia by preventing the formation of prostaglandin $E^2$, which sensitises peripheral pain receptors to stimulants.

Zomepirac is rapidly and completely absorbed following oral administration. After single dose of zomepirac, the onset of anagelsia occurs in about thirty minutes; the maximum effect is observed in one to two hours and the duration of action is four to six hours.

Although additional experience with the use of zomepirac in the management of pain in needed to determine its ultimate role, the results of clinical studies to date indicate that its range of effectiveness is broad. If patient obtain satisfactory relief with aspirin or acetaminophen, substitution of zomepirac is not needed. However, if pain is not controlled with these older drugs, or if they are not tolerated, zomepirac may be tried. It may also be used for moderate to severe pain in place of combinations containing narcotics for patients who can take oral medications.

Gastrointestinal reactions were reported most frequently and consisted of nausea, dyspepsia, gastrointestinal distress, abdominal pain, constipation, diarrhea, and vomiting. Other rare but reported effects include drowsiness, dizziness, and insominia. The drug should be used cautiously in patient with a history of peptic ulcers or gastrointestinal bleeding.

Because the drug is eliminated primarily by the kidney, patients with impaired renal function should be closely monitored, and it is advisable to assess renal function periodically during long term therapy.

Finally, it is noteworthy to mention again that on the basis of the results of numerous long term studies, it has been concluded that zomepirac does not produce psychic or physical dependence. □

The author would like to thank the Public Information Consultants (Eugene Kone) for sending the above information from the international association for the study of pain.

*Raymond Tong 8T3*

# Commonwealth Survey Could Cut Heart Attacks

A survey into the eating habits and lifestyle of the South Pacific islanders of Tonga which could lead to a breakthrough in the management of heart attacks and stroke in the Commonwealth has been suggested by a distinguished Indian doctor.

Professor Jacob Abraham's idea followed a visit he made to Tonga where he found the incidence of the killer diseases to be "remarkably low."

This despite the fact that the Tongans were hearty eaters, had a very high lipid content in their blood, were mostly overweight and they smoked, said Professor Abraham, a 50-year-old neurologist.

Professor Abraham, professor of neurosurgery and medical superintendent of the famous Christian Medical College and Hospital at Vellore in the southern Indian state of Tamil Nadu, added: "All these parameters (characteristics) represent potential risk factors in coronory heat disease and stroke."

"Blood lipid content among Tongans is very much higher than in Indians and Westerners. If they had the same degree of hyperlipidemia as the Tongans, the incidence of heart attacks and stroke among them would be much greater."

Is it, he wonders, because of the islanders' contented nature and

their "total lack of acquisitiveness" and a lifestyle devoid of the kind of pressures common to Western society, or is it the lack of salt and spices in their diet?

These are some of the questions he would like to have answered by his proposed survey, beginning with Tonga and followed by other Commonwealth countries in the Pacific.

Professor Abraham said: "If we find that it's not the salt and spices but just a question of tensions in society, then it's high time we started thinking very seriously about reducing those tensions. If, on the other hand, the lack of salt and spices in food is the factor, then we will have to recommend a reduction in their consumption, especially by families where heart attacks and stroke are common.

"They will simply have to make a choice between salt and spices and life."

Professor Abraham undertook the Tongan visit, which he combined with a trip to Malaysia, at the invitation of the Commonwealth Foundation.

The London - based Foundation, headed by Australian diplomat and playwright Ric Throssell, was established in 1966 by Commonwealth Heads of Government to promote closer co-operation between professional people within the Commonwealth.

Professor Abraham's observation on Tonga was based on his interviews with local doctors and health workers. He also saw a "paucity" of heart attack and stroke cases among hospital patients.

He said: "The survey could give additional insight into the etiology of heart attacks and stroke, and thereby lead to a breakthrough in the management of these diseases."

Although he did not visit the other Commonwealth islands in the region, he was sure conditions there were similar to those in Tonga. He thought that this phenomenon was well worth a proper study whose results could have beneficial effects for the rest of the Commonwealth — and beyond — and lead to a significant reduction in heart attacks and stroke.

"This sort of survey has never been done before. Tonga offers a natural laboratory, and we should take advantage of it. But we will need a major Commonwealth initiative to embark on it."

Professor Abraham suggested that the survey should be carried out by a team including doctors, health workers, para-medicals and statisticians. Members of the team should go from door-to-door to collect information. The data collected should be closely examined, and the team's recommendations widely circulated throughout the Commonwealth.

The idea has been welcomed by Professor Sir Kenneth Stuart, Director of the Medical Division of the Commonwealth Secretariat, the international organisation at the service of all Commonwealth governments. Sir Kenneth, from Barbados, himself a cardiologist who was previously Head of the Department of Medicine and Dean, Medical Faculty, University of the West Indies, says: "Heart attacks and stroke are now amongst the commonest causes of illness and death in developed and developing countries alike.


s-so-ci-a-tion:

**"A voluntary organization of persons joined together to pursue a common purpose or objective mutual to all."**

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"Additional information about them, particularly about their geographical distribution and environmental associations, is urgently needed. The proposed research should make a valuable contribution to a challenging field of medicine."

Professor Abraham's visit to Tonga has also resulted in the establishment of permanent links between his hospital and Vailo Hospital in the kingdom's capital, Nuku'alofa.

These links will be further strengthened when a young Tongan doctor, Dr. Palu Lasalo, visits Vellore in September at Professor Abraham's invitation. Dr. Lasalo, who is in his early 30s, will spend up to six months as a guest of the Indian hospital where he will receive special training in neurology. On his return, he will become the first Tongan doctor with knowledge of this highly specialised field — and one of the few in the South Pacific.

Professor Abraham, who was born in Kottayam in the southern Indian state of Kerala but who grew up in Calcutta, said: "I'm looking forward very much to Dr. Lasalo's visit. He will find that neurological conditions in India are similar to those in his own country. So the experience he gains here will be relevant to the needs of Tonga."

He added that the hospital would be pleased to welcome more doctors like Dr. Lasalo as well as nurses and other health workers for training, not only from Tonga but also from other Commonwealth developing countries.

Recalling his own trip to Tonga, he said: "It was a revelation. . . one of the best things to have happened to me. It was one of the most educative things that I have ever done."

His fondest memory is that of his meeting with King Taufa'ahau Tupou IV whom he presented with Nilgiri tea from south India. "I found the King to be extremely knowledgeable about the Commonwealth, particularly India, which he has visited two or three times. I was pleasantly surprised by his tremendous knowledge of India. . . he was even able to discuss the differences between Tamil and Malayalam!"

Tamil and Malayalam are two of India's major languages, mainly spoken in Tamil Nadu and neighbouring Kerala. Professor Abraham's mother tongue is Malayalam, and he also speaks Tamil.□

*by Asif Kahn*
*Vellore, India*
*August 1981*

(From *Commonwealth Feature*, Commonwealth Secretariat, London, England.)


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# A Glossus-In-Buccinator Survey Of Anatomical Names And Other *Bona Fide* Trivia

**Bill Nielsen 8T4**

*The student of a first anatomy course soon encounters and eventually adopts a vocabulary consisting largely of obscure words, hard to pronounce, not to mention the difficulty with discerning their meanings. This article attempts to enlighten both the anatomy student and the uninitiated alike as to the origin of a few anatomy terms—in doing so, the author takes us from the kitchens of Greece to the barnyards of Rome. Although this journey does not make the mastery of anatomy any less difficult, it does at identify some of the culprits from our ancient past at whom our frustrations may be directed.*

A first go at anatomy is sure to present today's student with a barrage of terms that twist the tongue and wrench the memory into a trembling wad of lumbrical jelly. Yet to the initiated, there appears beneath this impassive wall of ruinous verbiage an etymological foundation based on kitchenware and garden vegetables. This little corpus, dedicated to students of *bona fide* trivia, is written to elucidate some of that foundation by examining the derivation of a few anatomical words. I hope that this survey proves interesting and insightful to anyone undertaking the perilous journey of a first anatomy course, or the more perilous journey of repeating a first anatomy course.

I promised there would be kitchenware beneath the verbiage, and the number of human parts named after this genre of utensils may well indicate to which tables early anatomists turned to do their work. For example, the name of the arytenoid cartilage means "ladle-shaped." *Arytur* means ladle, and *-oid* is a Greek suffix that translateds "in the shape or form of.". This suffix is recognizable in all sorts of words, such as *asteroid* — "star-shaped.", *humanoid* — "in the form of a human," *discoid* —"in the form of a disc."; or *devoid* — "in the form of DEVO." The term *arachnoid*, or "spiderlike.", refers to a webby layer of tissue and has an interesting derivation. Arachne was a Lydian maiden skilled in the art of weaving who insulted the goddess Athene by producing a tapestry that not only outshone Athene's own skill at the loom, but depicted the errors of the gods in the woof. In anger, Athene destroyed it, and in fear Arachne hanged herself. Athene later felt ashamed at her own warped rage and restored the maiden to life, but as a spider destined to live as Arachne had died, at the end of a rope, still spinning and weaving.

The Greek suffix *-oid* is synonymous with the Latin suffix *-form*, which also finds its way into the jargon in words like *periform* (pear-shaped) or *pisiform* (pea-shaped). Back in the kitchen, the *acetabulum* of the os coxae is named for its resemblance to the Roman *acetabulum*, a small metal cup used to hold vinegar, and by this association with vinegar is kin to the word "acetic". Other utensils lend their shapes to anatomical vocabulary as well. Consider the Roman kitchen. On the shelves are found *platysma* and *patellae* (plates and saucers) and an occasional *pelvis* (basin) perhaps filled with *fabellae* (little beans). An *allantois* (sausage) hangs from the ceiling, and thanks to the slaving of the Greek cook, one can detect, wafting on the breeze, the scent of freshly baked *pancreases* and *placenta* (sweet bread and flat cakes) as they cool on the sill next to the *clavicle* (window latch). *Cribriform* indicates "in the shape of a sieve." and is a cognate of the modern cribbage board. As well, the anatomical position of the cribriform plate just inferior to the *crista galli* (cock's comb) may express the sentiments of the cool, intellectual anatomists concerning the cephalic development of roosters, and perhaps of sartorial strutters in general. Speaking of sartorial strutters, there is a connection between the name of the *sartorius* muscle and the sartors, or tailors, of days past. The sartorius flexes and laterally rotates the leg into the position that tailors used to assume at their benches, and has for that reason come to be known as the tailors' muscle).

Scores of other household items and implements of husbandry have also been immortalized by Roman anatomists who, unlike anatomists of subsequent ages, were too lazy to master an ancient, obscure, unpronounceable language to form

their words. From their barnyard come the *sella turcica, fossa, fovea, trochlea, falx,* and *malleolus;* which mean simply, a Turkish saddle, two ditches, a pulley, a scythe blade, and a little mallet, respectively. A *stapes* is a stirrup, a *frenulum* is a bridle, an *incus* is an anvil. The *radius* is a chariot wheel's spoke, broad and flat at the rim and narrow and rounded at the hub. *Zygous* means "yoked together", as in zygote or zygomatic process, and *azygous* means unyoked, referring to a vein that lacks a symmetrical twin.

From the sea comes the *hamatus,* or fishook, that names a peculiarly hooked bone in the hand. The *soleus* muscle, which is the broad, flat member of the triceps surae, is named after the fish we call sole. The *scapula* is an old term for an oar blade and the *naviculus,* observable under the sandle straps of the Romans, is so named because it looks like a "little ship."

Do not be fooled by the aforementioned nomenclature, however, into thinking that in Italy, all anatomy was studied by fishermen and farmers with a penchant for keeping their avocation barefoot and in the kitchen. In those days, dissection of human bodies was illegal; so naturally, study material was very hard to obtain.

To overcome this handicap, students developed an ingenious institution which became quite famous for its efficiency in procuring adequate numbers of specimens for anatomists. I am speaking, of course, of the Roman Legions, those proud dissecting classes that inspired the immortal Caesar (who was eventually studied by a class of senators) to comment, "*Omnis Gallus in tres partes dividatur.*" The many anatomical names related to the names of the tools of war testify that it was by following the legions of Germanicus that scholars became heirs to the windfall of early orthopedic experimental surgery. Many believe that the word *cadaver* itself comes from the Latin *cadere* meaning "to fall" in the sense of "those that fall in battle." Others think that it comes from the first letters of each word in the phrase *Carna data vermibus* which means "flesh given to worms." Knowing how anatomists work, I tend to believe that it was *cadaver* that was dissected and subsequently reassembled into the above phrase, but we may never know. Or care.

Anyway, with such a martial lot of dissectors in the open air labs that so liberally littered the countryside, it is not surprising that many human structures are named after weaponry. Macedonians traditionally fought with short swords that had curved pommels. That great arched artery, the *aorta,* resembles those curved hilts and by virtue of this resemblance perpetuates their name. *Xiphoid* means "sword-shaped", and *manubrium* refers to sword handles. *Thyroid* describes a cartilage shaped like the large shields carried by Minoan soldiers. *Sagittus* means "arrow", and thus names an arrow-shaped cranial suture (think of the lambdoid suture as the feathers). The *galea* was a helmet worn by Roman soldiers, protecting, of course, their galea aponeuroses. In Greek battles, the foot soldiers were arrayed in blocks, each block called a *phalanx,* and all the blocks were lined up in battle the way that *phalanges* are lined up in the fingers. The most famous Greek soldier of all was no doubt Achilles. An Achilles heel connotes a point of vulnerability and the tendo calcaneus happened to be this hero's weak spot because it was this spot that his mother, Thetis, grasped as she dipped the rest of the infant Achilles into the magical and protective waters of the River Styx. It appears that swiftness ran in the Achilles family.

Moving from soldiering to more refined subjects, the words of art and music also make contributions to the anatomical nomenclature. The *cricoid* cartilage, you may know, is named for its resemblance to a signet ring. The *fibula* is also named after a piece of jewelry, specifically, the fastening pin on the back of a brooch. This pin bears the same spatial relationship to the brooch that the fibula bears to the tibia. the *tibia* is itself a musical bone. It means "flute", for it was from the long, hollow shin bones of birds that flutes were once made. The name of the *coccyx,* or whistlebone, has a similar history. Because it looks like the beak of the cuckoo, the coccyx bears that name. The raven's beak also has an osseous namesake in the *coracoid* process. Strangely enough, there is even music in the term *cat-gut.* The "cat-gut" used in surgery does not come from cats, and never did. It has always been a product of sheep. Fiddle strings, which are very much like sutures, also come from sheep-gut, and a fiddle, centuries ago, was called a "kit"; the strings were known as *kit-gut,* a name that has evolved over the years to "cat-gut", perhaps because the sounds these instruments make led people to believe that there are cats about. However, this is likely the only relationship medical sutures have to feline bowel.

The cat's meow is as appropriate a note as any to end this litter-basket of facts on. In the future, if there are any specific words you want to know about but are afraid to ask about, or if you are just interesting names, and many more through a Latin-Canadian dictionary, or better yet, look through a copy of H.A. Skinner's *The Origin of Medical Terms.* Keep in mind that there are many more interesting names, and much more interesting parts of the body that were not discussed here, but have been christened relatively recently with interesting English names that are in vogue on playgrounds and at rugby parties. The ancients did this sort of christening too, and as a result, we resort to vulgar, everyday terms of Rome when we want to appear proper, or scholarly or precise. It is surprising the things that we anatomy students say when we really don't know what we are saying.□

# Little-Known Medical Facts

**Robert Hegele 8T1**

People ask me, "Rob, how is it that you are able to come up with these little-known medical facts?". To this, I usually proceed to examine the contents of my pockets with an air of signficance before responding, snobbishly, "I just make them up." Here then are a few of these facts for your information and immediate disposal.

### "Scarsdale Medical Diet is a Plagiarism"

Dr. Herman Turnover originally developed the Complete Carsdale Medical Diet in 1968. This diet consists of ingesting nothing but unlimited quantities of transmission fluid over a 2 week period. The weight loss from this method was remarkable; although many patients would unexplainably shift into reverse while walking down a sidewalk, leaving heelprints on the face of whoever was unfortunate enough to be behind them. Incidentally, Dr. Turnover met with an untimely demise in 1969, being bludgeoned to death with a '63 Plymouth's hubcap by his jealous ex-lover, Rita Slovenly, a part-time cashier at Manny's Self-Serve Texaco, after she found out that Turnover was secretly tanking up on Super Shell at the station across the street. □

### "Rat On"

The animal research of the legendary Irving Koven is known to all rat cutters far and wide. Of particular significance is his experiment in which he fed rats Roquefort cheese over a period of several months. These "guinea-pig" rats steadily developed a physical dependence on the euphoric and analgesic effects of Roquefort until finally mega-doses were required to satisfy their craving for the cheese. The rats had become, in effect, Roquefort addicts, their basic cellular physiology being altered to such an extent that they were biologically and psychologically unable to tolerate long periods of abstinence without going into "Roquefort-withdrawal" — a unique and well-defined syndrome consisting of pupillary dilatation, increased heartrate and profuse sweating, (the so-called "coldrat" state). The treatment of experimentally — induced Roquefort addiction in rats consisted of using a Roquefort analog or cogener (such as English Stilton or Danish Blue) and slowly decreasing the daily dose until total withdrawal was achieved. Unfortunately, this usually would have the effect of coverting a Roquefort addict into a Danish Blue addict, so the practical validity of this method of treatment has of late come into question.

Interestingly, a group in Denver has done extensive investigation into endogenous Roquefort-like compounds produced in the central nervous system of rats, the so-called "endoroques" and the receptors at which these hormones act. This is currently a promising area for future research. □

### "Go to Al"

Dr. Norbert Ross noted that over many years of intensive study, mice named "Al" were at a significantly higher risk of developing bladder cancer, whereas mice named "Humphrey" never developed cancer of any type. An audit of his 8 years study subsequently revealed that all 1124 mice used were named "Al" (and therefore none of the mice had the name "Humphrey"). Naturally, his results were invalidated; however, this was not done before the Panamanian government ordered a moratorium on naming newborn children "A1" (i.e. "Allan", "Allen", "Alan", "Algernon", etc.) and, by law, required *all* children (including females) to be named "Humphrey" to avoid the risk of contracting bladder cancer in the future. Therefore, all Panamanian infants born over a 7 month period were christened "Humphrey" and the sales of bibs and baby shoes with the initial "H" on them rose 100-fold during this time, forcing all the other initials into near bankruptcy. Consequently, extensive government aid was required to prevent massive layoffs at the individual bib factories. □

### "McBurney had a point"

Dr. James' "Oops" McTavish, a surgeon of some repute in Aberdeen in the late 19th century, was forced into early retirement because of a severe case of Parkinson's Disease which would tend to make his operative fields approximate Hiroshima to the casual observer. Once, while attempting to remove a colonic polyp, he uncontrollably punctured through the entire length of the colon in his inimitable bouncing fashion, causing it to bear a remarkable

resemblance to a large curved woodwind instrument. Towards the end of his career McTavish resorted to placing the scapel between his teeth for better control, but he abandoned this after he grew drowsy during a hysterectomy, with his head eventually ending up inside the patient's pelvis and McTavish himself snoring away quite perversely.

He is probably best remembered as the surgeon who described McTavish's Point, an infrequently used eponymn for the landmark located at the sixth left intercostal space in the anterior axillary line for evacuating a tension pneumothorax. One day while aiming his trochar at this point, he Parkinsonianly missed the intended site, puncturing instead the right abdominal wall at a point two-thirds of the way between the umbilicus and the anterior superior iliac spine, revealing an acutely inflamed appendix, which McTavish proceeded to suction through the trochar, causing the patient's viscera to decorate the surgeon's shoes. The patient, Isaac McBurney, needless to say, passed on to better things. But that point on his abdomen was named in his honour: McBurney's Point.□


# MEDICAL ALUMNI ASSOCIATION

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The Medical Alumni Association Student Aid Committee has a budget for 1981-82 of $72,550, to be used for scholarships, bursaries, Summer Student Fellowships, Transportation Fund grants, etc.

Loan funds are also available to both undergraduate and graduate students. However, there is a limit of $3,000 in loans to any one individual. Loans are interest free until the repayment date. After that, interest will be charged at prime plus 1%.

Scholarships, bursaries and summer fellowships are awarded through the Student Awards Committee of the Faculty of Medicine. Applications for bursary assistance can be made through Mrs. D. Alli, Student Affairs Office.

Further information and application forms can be obtained from Mrs. N.S. Shenstone, Room 3245, M.S.B., telephone 979-1539.

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**Editorial Note:**

The Toronto Medical Journal starts off a new season with this issue. Since we first published in 1923, our aim has been twofold: to convey information of academic and clinical interest to undergraduate medical students, and to act as a forum where the student can develop and refine his skills as a scientific writer. As well, we feature regular articles by guest clinicians and researchers, viewpoints, book reviews, and medical updates.

Today the Journal reaches about 1,000 medical students at the University of Toronto, as well as a number of U. of T. Medical alumni. In addition, many Canadian and foreign medical libraries are regular subscribers.

Naturally, if we are to continue to provide the high calibre writing our readers have come to expect, we require inputs from students at all levels. So we would like to take this opportunity to extend our invitation to all medical students to submit their articles, reviews, ideas and thoughts in time for our coming issue. Place them in the box marked "Medical Journal" in the Medical Society Office, or contact Davy Cheng 8T3 (923-6259). We publish four times a year, in November, January, March and April. The submission deadline for the next publication is November 20, 1981.

**Davy Cheng 8T3**